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DELAYED MASSIVE GANGRENE FOLLOWING HIGH TENSION ELECTRICAL BURNS

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The occurrence of massive gangrene days or weeks subsequent to a high-tension electrical burn is an interesting phenomenon reported in, but not explained by, the world literature. The purpose of this paper is to call attention to this phenomenon and to suggest a possible cause.

Delayed massive gangrene subsequent to electrical burns is reported [2-5, 11, 12, 16, 20-22] and is attributed by most authors to thrombosis of blood vessels.[1, 6, 7, 10, 13, 17-19] Jaffe[7] in 1928 reviewed a portion of the world literature. Jaffe[8] in 1929 was unable to reproduce experimentally the thrombi described in literature. Miller[15] felt that arteries do not become thrombosed beyond the involved necrotic area, but that veins do. Martin[14] stated that with supervening infection and the presence of thrombi, massive gangrene could occur.

VASOSPASTIC PHENOMENA

Vasospastic phenomena have been observed during and following electrical burns. This contraction of a vessel between electrodes was observed by Jaffe in 1929[8] and reported by Gaby.[5] Schnetz[20] details the case of a man who, six months after sustaining an electrical burn of the ring finger of the right hand, developed a Raynaud's phenomenon in this digit. Two years later gangrene of the distal phalanx of the ring finger of the right hand was present. Critchley[2] relates trophic changes including peripheral arterial spasm from autonomic involvement. Wakely[22] says that the edema of electrical burns is due to "... the transient functional changes in local blood vessels." Milliken[16] attempted "... in the hope that the vessels were constricted instead of thrombosed to relieve loss of arterial supply beyond the axillary artery, in a man with multiple burns of the right arm and forearm of delayed occurrence to make relaxing incision. The patient retained some function following this treatment." Kappelman[9] reports "... arterial rigor or spasm of arterioles."

I could find no reports of the use of sympathetic blocks in the treatment of delayed massive gangrene subsequent to electrical burns.

I have had one case which indicates that in some instances the delayed massive gangrene following electrical burns may be due to vasospasm.

CASE REPORT

A 34-year-old-man sustained a severe high-tension electrical burn of unknown voltage on August 15, 1957, and came under our care the next day, showing numerous electrical burns. There were third-degree burns of the left thumb, middle and little finger. A previously performed surgical amputation of the right index finger was noted. There was dry gangrene of the thumb. Dry gangrene of the skin of the right forearm extended from the middle of the radial aspect distally and was also present on the ulnar aspect at the juncture of the middle and distal thirds. The ulnar aspect of the right hand, some of the palm, and a major portion of the dorsum of the hand had viable skin. Small third-degree burns were noted on the left thigh and leg.

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Plastic coverage on the left upper extremity was carried out by Doctors Willard Schuessler and Donald Ewalt. A debridement of the right forearm was done on September 9, 1957.

Since it was felt that the right forearm could not be made functional a guillotine amputation at midforearm under tourniquet control was done on September 23, 1957. When the tourniquet was released, 45 minutes after application, the radial and ulnar arteries were isolated and ligated. Each was found to be the diameter of a small string. Dr. Donald Ewalt excised an area of granulations and applied a split thickness graft held with a stent.

The operation was concluded at 8:45 a.m. At 1:55 p.m., it became necessary for my associate, Dr. Robert Haan, to take the patient back to the operating room because of severe hemorrhage from the stump. At that time the radial and ulnar arteries were their usual size. In addition, an interosseus artery was visible and there were a number of small muscle bleeders. These vessels were ligated. Traction was reapplied. Healing was uneventful.

The miscroscopic examination of the blood vessels revealed disruptive changes in the walls of the arteries of the forearm. These changes were seen only with phase contrast illumination.

SUMMARY

One opinion of the phenomenon of delayed massive gangrene occurring days or weeks subsequent to high-tension electrical burns is that it is caused by thrombosis. It is difficult to understand how thrombosis can occur days or weeks subsequent to a burn unless infection is an element. The phenomenon of vasospasm following electrical burns has also been reported. As the arterial media in certain areas is destroyed by high-tension electric currents, I believe that perhaps in certain areas this media is sensitized by the passage of the current. Superimposed stimuli result in vasospasm, which in turn causes massive gangrene.

In cases of high-tension electrical burns, the patient should be carefully observed for prolonged periods of time. Should gangrene impend, sympathetic interruption should be carried out. Following an electrical burn blood vessels are abnormally friable; therefore, hemorrhage should be anticipated.

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AN EXPERIMENTAL ATTEMPT TO STIMULATE GROWTH BY A DISTRACTING FORCE ACROSS THE LOWER FEMORAL EPIPHYSIS

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Equalization of leg length in children by some satisfactory surgical or medical procedure has been a challenge since the earliest days of orthopedics Von Langenbeck,[1] in 1869, is credited with initiating the search for a safe, efficient and reliable method. Since then, the problem has been under constant experimental and clinical investigation.

The incidence of inequality of leg length has declined in recent years with the development of antibiotics and the Salk vaccine. These medical achievements have not touched the reservoir of problems secondary to congenital deformities, developmental aberrations, metabolic disturbances, tumors and trauma. Leg length equalization, therefore, still remains a basic orthopedic problem.

In the mid-nineteenth century (fig. 1), Hueter[2] and Volkmann[3] described the effects of pressure on epiphyseal growth. They noted that growth will be retarded whenever the normal pressures upon an epiphyseal line are increased for any reason such as unequal growth, improper body alignment, or muscle tension. Conversely growth will be accelerated whenever the epiphyseal pressures are decreased (fig. 2). This law furnished the essential basic information for the epiphyseal work of Haas,[4,5] Risser[6] and Blount.[7,8] The work of these men indicated the theoretical possibility of stimulating growth by applying a distracting force uniformly across an epiphyseal line (fig. 3). *A distracting force would decrease normal compression forces on the epiphyseal line and thus stimulate bone growth.*

The experiment reported here is designed to determine the effect of a distracting force applied across the growing epiphyseal line in experimental animals.

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METHOD

A. Device. An engineering firm was consulted and a stainless steel spring device was constructed (fig. 4), which would exert a maximum of 40 lb. of distraction force. The springs were sealed in stainless steel cylinders and the force was applied through 2 transfixing ⅛-in. pins. One pin was placed transversely through the distal femoral epiphysis and the other through the femoral shaft about 2½ in. proximally. Once the distracting force had been applied, it could be remeasured and adjusted while *in situ* by a spring tool. The size of the device was critical and had to be minimized to allow coverage with the tissues at the knee. The device was about 2½ in. long, the exact length varying with the amount of distraction and growth of the animal. The diameter of the outside cylinder was ½ in.

B. Animals. Experimental animals which satisfied the following criteria were necessary: (1) The young animals must have large enough femurs to hold the device; (2) there must be adequate future growth which would allow the device time to function; (3) young animals must be readily available, and (4) the animals must be easy to keep in the postoperative and convalescent period.

Sheep and pigs both seemed to satisfy these criteria and are readily available in our region. Sheep were first tried but proved unsatisfactory. Weaned pigs, weighing 40 to 80 lb., satisfied all criteria and proved to be excellent experimental animals.

A pig grows in height for 2 years. Therefore, the relationship of this growth period to that of the human is approximately 2:15. Based on this rough comparison, a 6-weeks' growth period in the pig would equal 45 weeks in the human, or 11 months.

C. Procedure. Ether anesthesia was used and the legs surgically prepared. The femoral condyles were exposed by a median parapatellar incision, dislocating the patella laterally. This

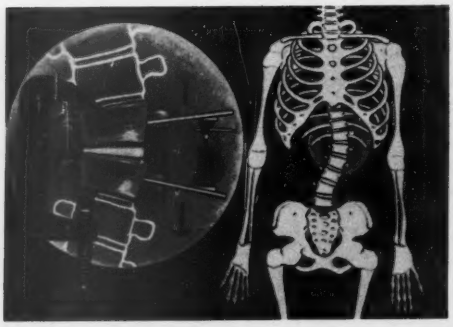
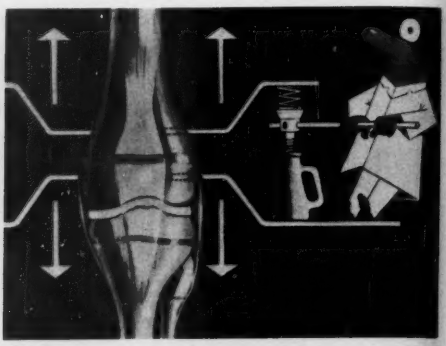


FIG. 1



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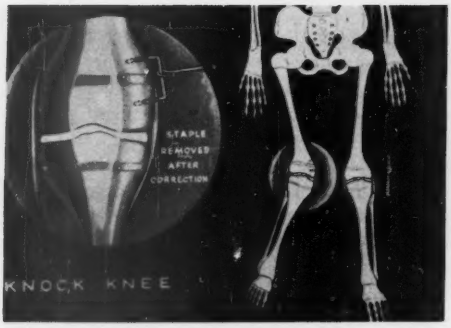

FIG. 2

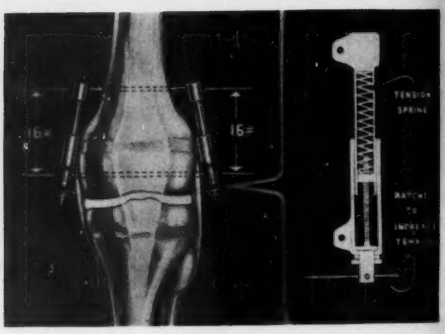

FIG. 4

allowed easy access to the condyles and the lower femoral shaft. A centering jig was then fastened to the femur with 2 cannulated screws gripping the condyles and 2, the femoral shaft. Drill holes through the condyles and shaft could then be made parallel and at an exact distance apart. The jig was removed and 2-⅛-in. Steinman pins were inserted. The distracting device was attached to the pins, which were threaded at each end, and the desired force created. Penicillin was dusted into the wound and intramuscular penicillin administered for 2 days. During this period, the pigs were kept in cages and then returned to the convalescent farm.

Upon recovery from the anesthetic, the pigs were ambulatory but usually remained non-weight-bearing on the operated leg throughout the experiment. This was considered to be an advantage, as the force of weight-bearing was eliminated from acting against the device and, in essence, simulated a nonfunctioning short leg. Soft tissue tension was created by the mechanism which resulted, in some instances, in skin erosion and infection. The operating time was gradually reduced from 1 hr. to 25 min. We discarded 8 pigs for the following reasons: preliminary testing of the device, errors in application and infection. There were 12 pigs, 9 experimental and 3 controls, finally accepted as presenting a true picture of the experiment.

Rapid growth, plus the loosening effect of pressure necrosis, made it essential, when possible, to operate at approximately 21-day intervals. Secondary surgical procedures, under general anesthesia, to re-establish the distracting force were performed through small medial and lateral incisions. The tension was adjusted by a small ratchet and measured by a spring-loaded tool. Fibrosis was always present around the device and to what extent this fibrosis decreased the effective force was impossible to ascertain.

RESULTS

Figure 5 shows the initial and final height of the animals at the shoulder and the original and

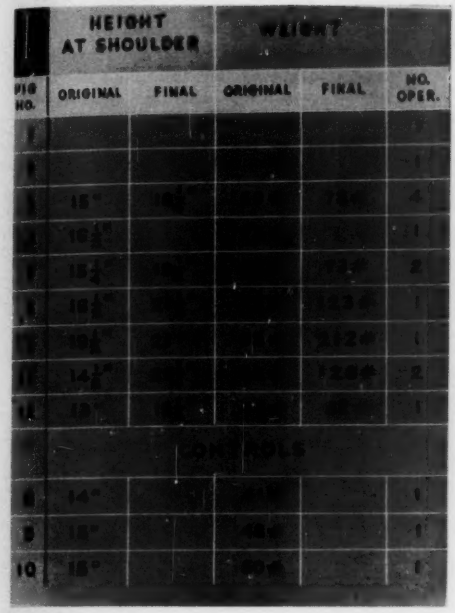

FIG. 5

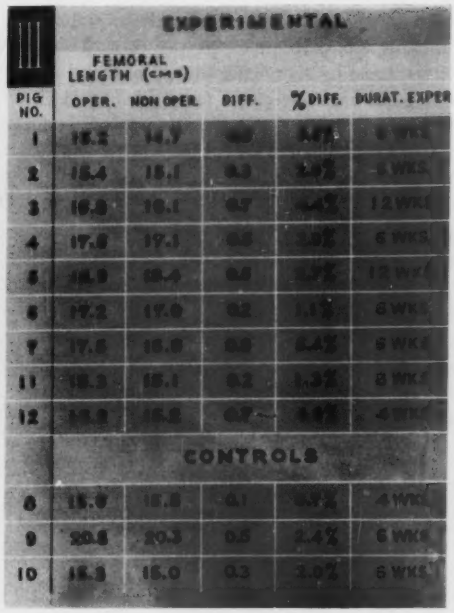

FIG. 7

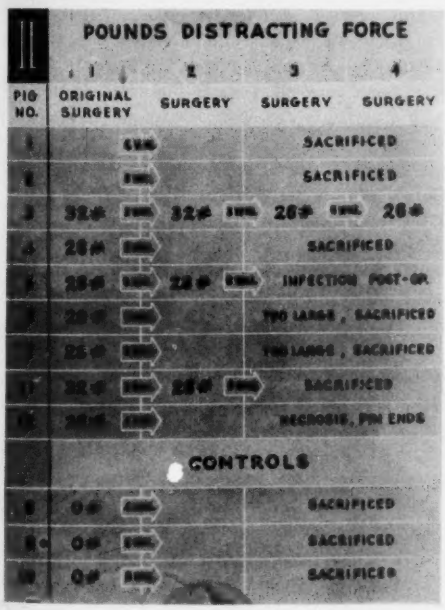

FIG. 6

final weights. The distracting force, intervals between procedures, and reason for termination of the experiment are given in figure 6.

The device was placed in 3 control animals without applying the distracting force. These animals were sacrificed 4 or 6 weeks later after 1 operation.

To ascertain the efficiency of the device and the amount of lengthening, the first 2 pigs were sacrificed after 6 weeks. The devices were loose and had lost their effective force. The decision was then made to operate at approximately 3-week intervals. Pig no. 3 was operated upon 4 times before he developed a local pin abscess. Pig no. 4 was sacrificed, inadvertently, by the animal custodian at 6 weeks. Pigs no. 5 and 11 were operated upon twice. The remaining pigs either became too large or developed abscesses and were sacrificed.

SUMMARY

Femoral growth, shown in figure 7, was not significantly altered by applying a powerful distracting force across the lower femoral epiphyseal line in the growing pig. The 3 control animals had essentially the same length stimulation, undoubtedly as a result of the surgical hyperemia.

The average gain in length of the experimental animals was 0.5 cm. or 3.1 per cent. This was determined by sacrificing the animals and measuring the exact length of the 2 femurs. Each of the 3 control animals had increased femoral growth, averaging 0.25 cm. or 1.3 per cent.

With this device, multiple operations were essential in the pig in order to maintain an effective distracting force. This experiment indicates that we have not found a practical means for clinical application of a distracting force across the epiphyseal line. The force of 32 lb., used in this experiment, far exceeds that utilized in treating juvenile orthopedic conditions such as fractures, dislocations, or infections. Therefore, one may assume that a comparable force inadvertently applied across a child's epiphyseal line does not disturb growth.

Further experimental work may be indicated to develop a distracting mechanism which does not require repeated reoperation.

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AN OBJECTIVE TEST FOR THE STUDY OF GASTRIC FUNCTION IN THE POSTOPERATIVE GASTRIC PATIENT*

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The role of surgery in the management of peptic ulcer and the criteria for selection of the surgical patient are well established. The type of surgery, however, is not as well defined. Recurrence rate and operative mortality have already excluded many procedures. It remains, however, for the less obvious sequelae of gastric surgery, namely disturbances in function as manifested by dumping, malnutrition and other postgastrectomy syndromes, to provide a guide to the proper operative procedure. Not only does the study of these sequelae aid in selecting the best operative procedure but also provides greater understanding of gastric physiology.

Although many postgastrectomy syndromes have been recognized, the dumping phenomenon is perhaps the most common and has been the subject of the most study. In our clinic we have defined dumping as a condition occurring soon after eating, usually a meal high in carbohydrate, which is typically manifested by sweating, unpleasant warmth, flushing, nausea, palpitation and even syncope. It may be associated with epigastric fullness or cramps, vomiting or diarrhea. It is usually of short duration and may be partially relieved by lying down. Although the reported incidence of this syndrome varies, careful follow-up in our own clinic demonstrated 1 per cent severe, 21 per cent moderate, and 22 per cent bothered by one particular food. Thus, 2 in 5 have some or all of these symptoms postoperatively. The dumping syndrome is therefore an important aspect of gastric surgery.

Roberts and associates[7] in 1954 demonstrated that the dumping symptoms were closely correlated with a loss of plasma volume, presumably into the intestinal tract. They were able to duplicate these symptoms using a test meal of glucose solution, and concomitantly measure the change in plasma volume by the Evans blue technique.[4] They concluded that the ingestion of hypertonic solutions caused a loss of plasma from the vascular system into the intestinal tract, and that this disparity between the vascular system and its volume produced the dumping symptoms. We concur with these authors, and have employed a modification of their experiment in evaluating patients after gastric surgery. We feel that the test, as modified, provides an objective method of identifying those patients who have an actual disturbance in gastric function rather than a psychic disturbance, and further provides to a certain extent an objective means of evaluating different types of gastric surgery.

METHOD

Radioactive iodinated serum albumin (RISA) has been employed to measure plasma volume rather than Evans blue method as originally described. With the patient in a fasting state, a standard injection of RISA is administered, and 3 serial blood samples are drawn at 10-min. intervals to determine the disappearance curve of the RISA. The patient then ingests 75 gm. of glucose in 240 cc. of water. With the patient recumbent, serial blood samples are drawn at 10-min. intervals for a period of 1 hr., postingestion. The plasma volume at each time interval is determined by standard technique, and by comparison with the plasma volume determined before the ingestion of the glucose, the amount of shift established. The amount in cc. representing the greatest shift, divided by the total plasma volume and converted to per cent gives a figure termed "per cent maximum plasma shift." This figure is felt to be more representative than a figure expressed in cubic centimeters, since it makes allowance for body weight differences.

RESULTS

Over the past 3 years, 107 patients have been studied by this method, and since more than 1 test was performed in many instances, a total of 134 studies has been carried out. The amount of

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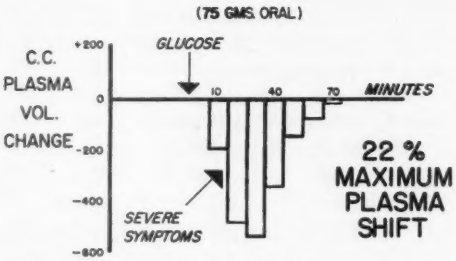

FIG. 1. Typical plasma shift in a patient with severe symptoms.

glucose was not changed even though we encountered explosive diarrhea in several instances, a problem previously mentioned by Roberts and Randall. This was the only difficulty encountered during the various tests.

Immediately following the ingestion of the glucose there was little or no loss in plasma volume (fig. 1). Usually by the 20-min. specimen, however, there was a marked loss in volume which peaked with the 20- or 30-min. sample and then returned to preglucose volume by the last or 60-min. specimen. Although in some instances the peak was later, the curves were fairly constant and were not only consistent from patient to patient but were consistent with the same patient. Figure 1 is a typical curve for a patient with a marked shift and with dumping symptoms.

Initially the test was used to identify those patients in which there was an actual physiologic disturbance as the basis for their symptoms. Controls were selected from either preoperative gastric patients or patients with other diseases. None of these had dumping symptoms. On the basis of a scattergraph using all patients studied, a working level of 10 per cent shift was established as a dividing line between dumpers and non-dumpers (fig. 2). None of the controls showed a shift of 10 per cent and the majority showed no shift at all or one of less than 4 per cent. In the patients with previous gastric surgery but with no symptoms demonstrable after repeated questioning, 87.5 per cent shifted less than 10 per cent. Conversely, in those patients who complained of dumping symptoms, 85 per cent showed a plasma shift of 10 per cent or greater, and one patient showed a loss of 37 per cent. Thus as a diagnostic test, we found that there was little or no change in the controls and that by using 10 per cent shift as an arbitrary dividing line, 1 in 8 gave a false positive and 1 in 6 gave a false negative. This was felt to be fairly close correlation for a clinical test, especially since no test had existed before.

After the establishment of the usefulness of the test, the subjects were grouped according to the type of surgery performed: (1) none; (2) posterior gastroenterostomy; (3) gastrectomy with Billroth II anastomosis; and (4) gastrectomy with Billroth I anastomosis plus vagotomy. The latter group was further broken down as to extent of resection, 50 to 60 per cent and greater, and also on the basis of size of the anastomotic stoma, regular or small. By employing the average plasma shift for each of these various groups (fig. 3), all operated patients were found to be above the average of the controls. The largest shifts were found in the group with gastroenterostomies and the smallest were in the 50 to 60 per cent gastrectomy group who had a Billroth I anastomosis, vagotomy and small stoma. Further, if each group were divided into patients with dumping and those without dumping, (fig. 4), the average shift of those with symptoms were very similar in all groups except those with the small stoma. Also, the levels of those with no symptoms were remarkably constant in all groups.

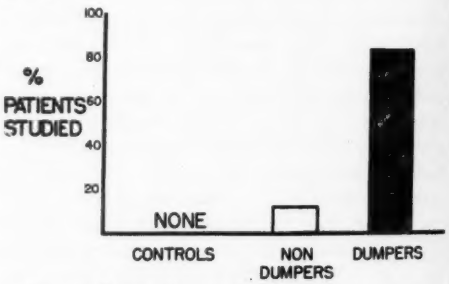

FIG. 2. Correlation between dumping symptoms and 10 per cent plasma shift.

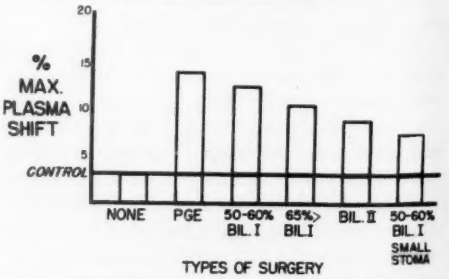

FIG. 3. Average maximum plasma shift with various operative procedures.

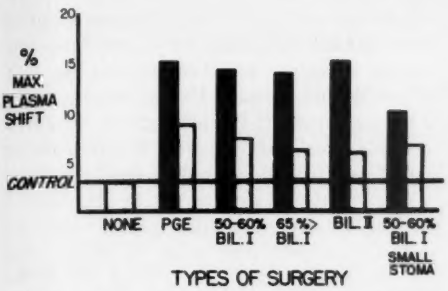

FIG. 4. Average maximum plasma shift in dumpers and non-dumpers with various operative procedures. ■, average, dumper; □, average, non-dumper.

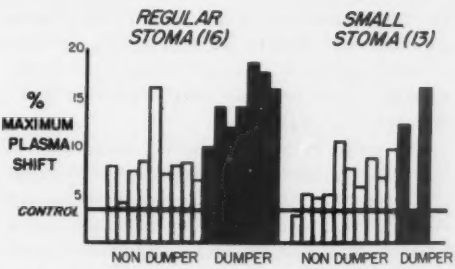

FIG. 6. Comparison of plasma shift between patients with a large stoma and patients with a small stoma.

DISCUSSION

As previously noted, we have employed this test in 107 patients for a total of 132 investigations. It is obvious (fig. 2) that an unoperated or intact gastric mechanism does not respond to a test meal of hypertonic solution either by dumping symptoms or by loss in circulating plasma. When gastric surgery has been performed, however, and even in the absence of dumping symptoms, there is a marked shift in plasma, and when dumping symptoms are present, this usually exceeds 10 per cent of the total plasma volume. On the basis of this, 10 per cent was selected as an arbitrary dividing line for the establishment of a positive or negative result. A small number of non-dumpers shifted greater than 10 per cent. It might be conjectured that these represent individuals with a very stable nervous system which responds minimally to large fluctuations in the vascular system. Conversely, the patients with symptoms but with no demonstrable shift may possess a fairly sensitive nervous system which responds maximally to small shifts in the vascular system. Also, other mechanisms, as suggested by Wangensteen and co-workers[6] may exist which would account for the symptoms.

In using the test to evaluate the different types of surgery, we have expected some change with all types. In spite of our previous experience with gastroenterostomy[8] which indicated that this type of surgery gave the least nutritional disturbance, this group showed the greatest shift. Perhaps much of this can be explained by the fact that these were patients done several years ago before the importance of correct placement and size of the anastomosis was appreciated.[2, 3]

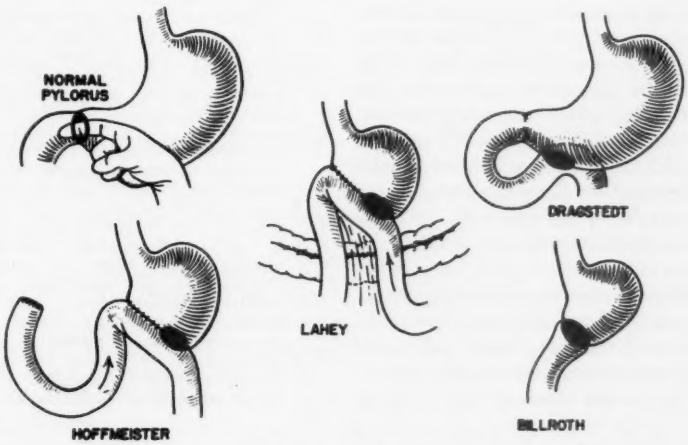

FIG. 5. Concepts as to size of the anastomotic stoma.

Further, a large percentage of these patient were admitted and studied because of the severity of their symptoms. Thus, our results bear out the validity of the test, but do not necessarily incriminate the procedure.

The group of patients having some type of Billroth II procedure also represented an older group of patients since, in recent years, the Billroth I operation has been emphasized in this clinic. It is well known that dumping symptoms tend to diminish with time. This may explain the smaller shift of this group. Whether this diminution in symptoms represents an accommodation on the part of the individual, *i.e.*, "learning to live with his disease," or an actual return to more nearly normal physiology is not known, yet the test might seem to indicate a restoration of more nearly normal physiology.

In the groups in which a Billroth I type of anastomosis was performed, the averages varied. We expected that the group with a 65 per cent or greater resection would show more shift than a lesser resection, but this was not true. Here again, the elapsed time factor may enter in, since it has been our more recent policy to perform 50 to 60 per cent resection and those of 65 per cent or greater are therefore a much older group. We were discouraged, however, to find that the Billroth I group of patients showed just as much plasma shift, and even though an older group, the Billroth II showed slightly less.

Because of these findings, our standard gastric procedure (50 to 60 per cent gastrectomy with Billroth I anastomosis plus vagotomy) was modified by creating a smaller anastomotic stoma. This stoma measured 1 to 1½ cm. in diameter or roughly the size of the surgeon's finger. This concept, that the normal pylorus is this size (fig. 5), is not new since it has already been reiterated by previous gastric surgeons, Billroth and Hoffmeister in former times and more recently Lahey and Camblos[5] and Dragstedt.[2] Thus, in the past year, a group of patients with the small stoma was developed. Despite the fact that this was the newest or more recent group of patients, only a few patients demonstrated dumping symptoms. This group was compared not only with the previous groups of patients (figs. 3 and 4) but also with the group who had had similar surgery but no effort to control stomal size (fig. 6). Thus, even though the small stoma group was the more recent and has potentially less chance to accommodate, it showed less dumping and less shift than all the other groups. Further, in comparison to the regular stoma-Billroth I group, the difference was even more marked. Recent work by Abbott and others[1] suggests a similar pattern.

SUMMARY

From the material accumulated in this study, both by laboratory test and by clinical evaluation, we have formed the following conclusions:

1. An objective test is available for the identification of the actual dumping patient.

2. A plasma shift of 10 per cent or greater correlates very closely with the patients clinically identified as "dumpers."

3. This test may be used in the evaluation of various types of gastric surgery.

4. The creation of a small anastomotic stoma after resection appears to give a better functional and objectively measurable result.

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THE ELDERLY THYROTOXIC PATIENT

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Someone has aptly labeled the thyroid gland as "the lady of the endocrine system," and for centuries the mysteries and intrigues she has held within her grasp have charmed and outwitted the medical profession. Her many and varied personalities as manifested in primary hyperthyroidism, the solitary adenoma, carcinoma, hypothyroidism and the adenomatous gland with superimposed toxicity have led us a merry chase for years. To some she has given bits of knowledge which have benefited us all, but there is no one she has confided in completely. Each one treating diseases of this gland has some aspect that interests him most. To me the problems arising in the elderly individual with thyrotoxicosis are especially intriguing.

In the main, hyperthyroidism is a disease of the early and middle ages but it is becoming increasingly apparent that the elderly individual can be and is affected. A study at the Lahey Clinic in Boston in 1954[1] showed that 12 per cent of their cases of hyperthyrodism were over 60 years of age. Of this group 80 per cent were women, as is true in the younger group. However, in this older group approximately 60 per cent showed adenomatous hyperplasia with superimposed toxicity rather than the primary hyperplasia of Graves' disease. Since most cases of primary hyperplasia have responded well to radioactive iodine, I will confine my remarks to those with the adenomatous hyperplasia. The increasing incidence of toxicity in the nodular goiter causing hyperthyrodism in our long-living population is another reason for our advising thyroidectomy in these glands earlier in life.

That hyperthyroidism in the elderly individual is quite a paradox as compared to the younger ones, is obvious in these pictures (fig. 1). The onset is insidious and often the diagnosis is not made for many years. The symptoms and signs are so gradual that even the patient may be led to consider them as "just old age." Dr. George Crile, Jr.,[7] observed many years ago that "in the young, hyperthyroidism expresses itself as stimulation and in the old as exhaustion." In the younger groups the average lapse of time before diagnosis is 6 to 7 months, but in the elderly thyrotoxic, it is frequently 5 to 10 years. In contradistinction to the younger group, these people are quite apathetic; indeed, they were called "apathetic hyperthyroids" by Lahey and Hoover[8] many years ago. They experience a definite weight loss and frequently a marked myoatrophy producing weakness and easy fatigability. There is anorexia but again it is so insidious at onset that it is frequently overlooked. There is no definite temperature intolerance, yet they may be seen wearing heavy clothes far into the summer—again, another manifestation of "old age." There is no hand tremor but frequently their hands are unusually warm. This "warm hand" is considered a common sign and one for which to look. Rarely is there any widening of the palpebral fissure producing exopthalamus. Then, too, physically the thyroid gland in the young shows primary hyperplasia, whereas in the older group there is nodular hyperplasia. Cardiac complications are unusual in the young and common in the old. The diagnosis in the young is clear cut generally, but in the elderly it may be quite obscure.

The clinical signs unique to this group and, in particular those with the nodular goiter, are the ones related to the cardiovascular system. This actually is not unreasonable for it is well documented that the cardiac output is increased in direct ratio to the increase in oxygen demand. Other factors in the increased cardiac activity in hyperthyroidism, as pointed out by Bartels,[2] include: (1) abnormal irritability of the accelerator nerves of the heart; (2) irritability of the heart muscle due to excess circulating thyroxin, and (3) myasthenic effect of thyroxin on the heart muscle similar to that on skeletal muscle fibers.

The presence of any sign of cardiac malfunction, such as palpitation, irregular rhythm, or increased pulse rate in a patient with an adenomatous goiter should make one suspicious of toxicity. The two most common cardiac signs are auricular fibrillation and congestive failure. In this group these manifestations have been found

FIG. 1. Hyperthyroidism in old and young. (From Surgery, Gynecology and Obstetrics)

in 32 per cent and 15 per cent of the cases respectively. Interestingly enough, even far advanced cardiac problems due to thyrotoxicity in this group will revert to normal after removal of the gland. Physically these people are asthenic, rather fragile in appearance and in the majority the goiter can be seen or palpated. However, those with primary hyperplasia may have a gland too small to palpate.

DIAGNOSIS

Here, as in most diseases, diagnosis is made on a careful history and physical examination. Suspicions may be confirmed by a determination of the protein-bound iodine content of the peripheral blood. This has proved to be an accurate and efficient method for determining thyroid function. I^{131} uptake studies have been unnecessary in our experience.

MANAGEMENT

In an effort to obtain the current nationwide feelings regarding the problem of management of toxic nodular goiter in the elderly individual, we contacted 100 members of the American Goiter Society for their personal opinions. Obviously, because each case of this type must be individualized, this questionnaire concerned the basic pattern of therapy. In the literature, the pendulum has seemingly swung, even in this group, to radioactive iodine, but this poll revealed interesting findings (fig. 2). We received 80 answers and 66, or 82 per cent, of this group—which is composed of both internists and surgeons—still believe subtotal thyroidectomy to be the procedure of choice in these cases. Of these men, 59 per cent use propylthiouracil and iodine before surgery; 6 per cent use Tapazole, and 11 per cent iodine alone. For control, 16 per cent use radioactive iodine either initially or after propylthiouracil. All, however, agreed that, in the rare patient who cannot safely tolerate surgery, but has a good radioactive iodine uptake, the I^{131} should be used.[2] It is interesting to observe also that 85 per cent of these men use general anesthesia and 15 per cent use local anesthesia. Thus, then, the popular method of treating these patients is still surgical and the reasons given by this cross sectional group are as follows. (1) It is safe. The mortality rate is less

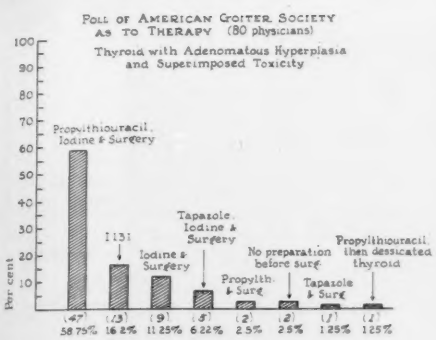

FIG. 2

than 0.5 per cent. (2) It is expedient. None of the uncertainties of I^{131} are encountered. (4) It is more economical. As one knows, in this group, because of the poor uptake of I^{131}, it may be necessary to repeat it, thus increasing the expense. (5) It makes possible a pathologic study of the removed gland. Carcinoma can and has occurred in toxic nodular goiters. (6) It relieves tracheal obstruction which occurs in 60 per cent of these patients.

It, too, is our opinion that these people with adenomatous hyperplasia with superimposed toxicity can only be treated adequately by surgical removal of the goiter. Radioactive iodine uptake in these nodular goiters is unpredictable, therefore calculation of dosage cannot be ascertained with accuracy, which results in higher incidence of incompletely controlled toxicity. This nodularity increases the time involved in controlling these people before surgery, even with propylthiouracil, but we have found that all can be controlled by time and increasing dosage. We are using increased dosages of propylthiouracil up to 1000 to 2000 mg. daily without evidence of increased toxicity from the drug and with a definite decrease in the time interval to produce euthyroidism. We have had no problems with agranulocytosis using this larger dose. For 14 days before surgery we use Lugol's solution, which seems definitely to facilitate the operative procedure. It was interesting to note in our poll of the American Goiter Society that many of the midwestern surgeons, including those at the Mayo Clinic, use only 5 to 7 days preparation with iodine solution in the milder cases. Some few others use no preparation at all. I think it significant that these men, working where this type of goiter has been most frequent over the years and the experience has been greatest, have found it unnecessary to control the milder cases in this group. However, it has been our practice to reach a state of euthyroidism on all cases, thus minimizing the possibilities of postoperative complications which this age group tolerates so poorly.

Those with thyrocardiac disease require the most conscientious preparation. It has been found that reaching a euthyroid state is of even more importance than the cardiac drugs, such as digitalis and the diuretics. Thus the use of propylthiouracil or Tapazole takes precedence over these cardiac drugs. Indeed, digitalis seems to be ineffective in these people until the thyroid function is decreased to normal.

In addition to the above, we have found local anesthesia and tracheostomy to be of great value in this group. With minimal preoperative medications, such as 50 mg. of Demerol and $\frac{1}{250}$ gr. of atropine, a careful infiltration of the anterior cervical region was carried out as described by Crile[7] (fig. 3). The skin at point 1 is given a firm pinch and immediately, using a 25-gauge needle, the anesthetic solution is injected. By slowly and carefully following the numbered areas, the neck can be effectively anesthetized with surprisingly little pain to the patient. To older surgeons, this may seem very elementary, but it is surprising how many of us have never done a thyroidectomy under local anesthesia. It is our custom to use from 90 to 100 cc. of 1 per cent Novocaine and then massage the tissues thoroughly for uniform penetration. The prethyroid and sternocleidomastoid muscles are also injected when exposed. This, with an occasional intravenous injection of 25 to 50 mg. Demerol, but never over 100 mg., has proved to be the ideal method for us. Postoperatively all anesthetic pulmonary complications are absent, as well as the increased cardiac stress of the general anesthetic. We believe that the efficiency of local anesthetics has increased since they were generally abandoned for this operation, and that they should be re-evaluated for use in this particular group of people. Tracheostomy adds no more morbidity to the procedure and decreases the cardiac burden for this elderly group. The respiratory exchange is greatly facilitated and the bronchial tree can be kept clear by the patient with a minimum of effort, thus decreasing the possibility of post-

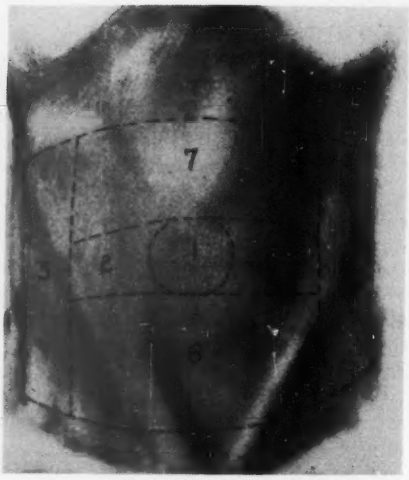

FIG. 3. Local infiltration for thyroid surgery[7]

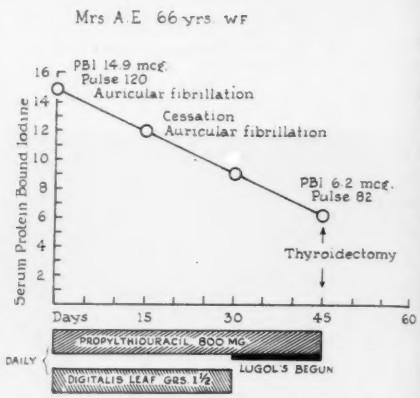

FIG. 4

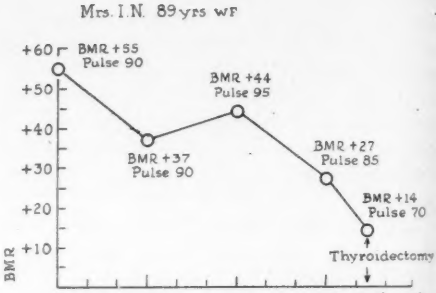

FIG. 5

operative pneumonia and anoxia. Experience has taught us that these people tolerate anoxia quite poorly. We have found that both the local anesthesia and the tracheostomy are well tolerated by the patient, but that a few moments should be taken before surgery to explain to them the advantages of each. The tracheostomy tube is removed after 3 to 4 days with prompt healing of the entire wound. We have found that these people have very poor drug tolerance and require only minimal postoperative medications. Two brief case reports are included to point up some of the problems encountered in these elderly individuals (fig. 4).

CASE REPORTS

Case 1. Mrs. A. E., a 66-year-old white woman presented with a 1-year history of increasing nervousness, easy fatigability and 10-lb. weight loss. She was admitted as an emergency after an acute onset of auricular fibrillation which responded to rapid digitalization. Physical examination on this admission revealed a nodular enlargement of the right lobe of the thyroid with a pulse rate persistently at 120 per min. Her protein-bound iodine determination was 14.9 μg.

Propylthiouracil, 800 mg. daily, was begun with alleviation of all symptoms in 30 days. The digitalis was discontinued at this time and the protein-bound iodine determination was 6.2 μg. After 14 days of preparation with Lugol's solution, a right total lobectomy was done under local anesthesia and followed by tracheostomy. The tracheostomy tube was removed in 4 days and the patient experienced an uneventful postoperative course. It has now been 2 years since surgery and there is no evidence of cardiac disease. This patient represents a typical toxic nodular goiter with cardiac complications. She responsed quite well to the antithyroid drugs and the surgery.

Case 2. Mrs. I. N., an 89-year-old white woman, (fig. 5), was admitted to the hospital with recurrent uncontrollable diarrhea of 8 years' duration, with easy fatigability, nervousness and increasing heat intolerance. Physical examination revealed nodular enlargement of the thyroid with marked tracheal obstruction; her basal metabolic rate (BMR) initially was +55. She was given 800 mg. of propylthiouracil with a decrease in her BMR to +37 within 30

FIG. 6. Size of gland and tracheal obstruction. Case 2

days. However, after another 30 days of the same dosage her BMR went back up to +44 and the propylthiouracil was increased to 1000 mg. daily. At the expiration of 30 days, 90 days total treatment time, her BMR was +27 and there were objective signs of tracheal obstruction. Figure 6 shows the amount of tracheal obstruction and size of her gland. The propylthiouracil was then increased to 1500 mg. daily and Lugol's solution was begun. In 14 days, a total of 104 treatment days, her BMR was +14. All of her symptoms had been relieved and her diarrhea had been absent since the first month of therapy. Subtotal thyroidectomy was carried out under local anesthesia, followed by tracheotomy without difficulty. The tracheotomy tube was removed in 4 days and she had an uneventful postoperative couse.

All symptoms, including the diarrhea, were completely relieved postoperatively. The patient lived to the age of 93, when she died from bronchial pneumonia. This patient exhibited quite well the difficulties encountered with propylthiouracil in the toxic nodular goiter. For reasons that we were not able to explain, during her second month of therapy under 800 mg. of propylthiouracil, her basal metabolic rate went up to +44. Because of her increasing tracheal obstruction we believed it imperative that she reach euthyroid state; therefore 1000 mg. and then 1500 mg. of propylthiouracil were used daily so that she rapidly reduced to normal thyroid function. It is our belief that all cases such as this can be controlled by time and increasing the dosage of propylthiouracil. We have not found it possible to produce hypothyroidism with this increased dosage.

SUMMARY

The toxic nodular goiter in elderly individuals remains an intriguing phase of thyroid disease. By careful preparation and surgical care, it can be controlled and removed successfully with lasting results.

Adenomatous goiter with superimposed toxicity is an increasing problem with our increased longevity. This superimposed toxicity in adenomatous goiter is another reason for their removal at an early date. By carefully controlling the patient before surgery, treatment can be successful. All patients of this group can be brought to euthyroidism by time and increasing antithyroid dosage. Radioactive iodine is not the therapy of choice except in poor risk patients and those with primary hyperplasia with toxicity. Local anesthesia and tracheotomy are of great benefit in these people.

Acknowledgment. The interest and cooperation shown by the members of the American Goiter Society in this study is appreciated.

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THE PROBLEM OF METABOLIC THERAPY IN MAJOR BURNS

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Despite many recent improvements in emergency treatment of trauma, little has been added to the therapeutic regimen for thermal burns. Moyer[8] has stated that the mortality in major burns has not changed in the past 100 years. In 93 cases of burns over 20 per cent of body surface, Clark and Hanson[4] recently found a mortality of 40 per cent. Bull and Fisher[3] further observed that in medium sized burns the prognosis has not improved in 10 years. In the patient over 60 years of age, Blocker[2] has not seen 1 survivor who had more than 30 per cent body surface burns.

Much of what is administered as parenteral solution to replace the depleted body fluids in the major burn is based on calculated formulas for each successive 24-hr. period. The formula of Evans and Bigger[5] is generally employed. Based on estimates of per cent of body surface burned, and total body weight, the entire parenteral fluid replacement for each successive day is systematically calculated and administered. It is generally recognized that such replacement formulas are inadequate for burns greater than 50 per cent.

Jackson[7] has noted that in a severe burn ½ L. of plasma may be lost per hr. He is disdainful of the use of replacement formulas except for large groups of patients in a general emergency. He prefers to individualize treatment of the basis of blood volume, hematocrit, hourly urine output, and clinical findings (blood pressure, color, restlessness, thirst, temperature, and sensorium reactions). It is further maintained that treatment by formulas may differ 100 per cent from that based on the individual clinical requirements.

The problem of how to manage parenteral therapy in a burn covering 30 to 50 per cent of body surface is thus a controversial issue. The purpose of this study is to evaluate this situation and provide rational recommendations for improving patient care and survival.

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MATERIALS AND METHODS

Case records of all adult patients with thermal burns greater than 30 per cent of body surface at the Johns Hopkins Hospital, 1953 to 1958, were reviewed. Particular emphasis was placed on total daily fluid intake, urinary output, clinical course and common pathologic findings, if any.

All patients selected for review were those who were seen within hours of sustained injury, and treated a minimum of 48 hr. The age range was 16 to 83 years and the average weight, 60 to 80 kg.

RESULTS

There were 27 adults with major burns treated (table 1). In general the group may be divided into 2 categories: those with burns 30 to 40 per cent, and those with burns greater than 50 per cent.

Patients with burns 30 to 40 per cent were all treated with the standard Evans[5] formula. Those with greater than 50 per cent burns were managed in a variety of ways.

In 30 to 40 per cent burns, a 33 per cent mortality was observed. In 22 per cent of those cases terminating fatally, renal shut-down and anuria were present.

In 50 per cent burns, an unsurprising 88 per cent mortality, which in 87 per cent of instances was complicated by renal shut-down, was noted.

A triad of pathologic findings (table 2) were noted in the 14 cases autopsied. Generalized tubular hemosiderosis and necrosis (93 per cent), marked adrenal lipid depletion and hemorrhage (64 per cent) and central lobular hepatic necrosis (36 per cent), were commonly observed. No conclusive reason for the hepatic pathology was derived.

DISCUSSION

At face value, this series of burns is no different from a host of others previously documented.[3, 4] There is no essential change in survival rate in either group.

TABLE 1
Clinical findings

Per Cent Burn	Number Patients	Mortality		Persistent Acid Urine	Renal Shut-down
		No.	Per cent		
30 to 40	18	6	33	7	4
50+	9	8	88	8	7

Table 2
Pathologic findings, 14 autopsies

Findings	No.	Per Cent
Significant adrenal lipid depletion...	9	64
Generalized tubular hemosiderosis and necrosis.....	13	93
Central hepatic necrosis.....	5	36

All physicians[6] are generally aware of the problems of hypo- and hypernatremia and kalemia in the burn patient, and adequately replace these losses. However, adequate colloid replacement is often underestimated when based on formula alone. Frequent hematocrit determinations during the first 24 hr. decreases this problem. In the 30 to 40 per cent burn group, 2 of the 4 deaths which occurred within the first 48 hr. had developed hematocrit values over 60. These patients were given inadequate colloid, despite previous calculations. Both patients were oliguric until death and probably died as a result of hypovolemia.

When one analyzes strictly on a volumetric basis the fluid therapy administered to the 30 to 40 per cent burn patients, a striking feature is observed (fig. 1). In general, all were managed on Evans' formula. In some instances, patients were allowed oral intake which resulted in an unexpected increase in total fluid intake. The patients treated strictly on a formula basis all had low hourly and total urine excretion. A linear increase in total output was observed in those receiving more than the formula. From this one can conclude that the strict formula, even where applicable, generally does not supply enough total volume to maintain a satisfactory urine output.

There are distinct advantages in maintaining high urine flow in the initial burn phases. Olson[9] has found a distinct correlation between the degree of hemoglobinuria and the duration of the anuria. By providing adequate volemic replacement one of the factors in acute renal failure could thus be avoided. It has long been known[1] that hemoglobin precipitates readily in acid medium. The observation[1] that patients in severe postburn renal shut-down had had prior

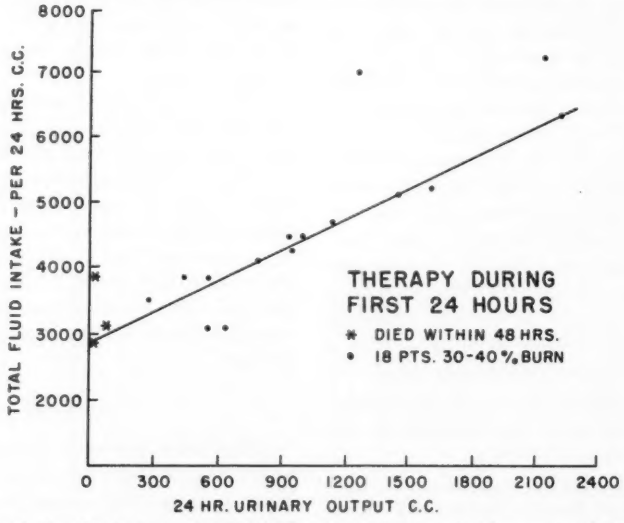

FIG. 1. Linear relationship between total initial volume given and urine output in 30 to 40 per cent burn patients in the first 24 hr. postburn.

persistent acid urines may indicate a relationship. The further recommendation[11] that increasing sodium bicarbonate or electrolyte solution would encourage excretion of an alkaline urine seems reasonable in light of these facts. Of 15 patients in this entire group with persistent initial acid urines, 14 died; 11 had demonstrated renal shut-down to a marked degree. Increasing early urine output also promotes early elimination of other toxic burn products. There is, of course, a reasonable physiologic limit to increased urine output and fluid intake.

The problem of increasing total volume of parenteral fluid intake early in major burns is not just a matter of ordering more solution. All parenteral fluids take time in administration, and particular emphasis must be placed on colloid replacement. Others[6, 10] have long championed massive oral electrolyte therapy in burns. The obvious problem of vomiting is occasionally avoided by not including plain water, orange juice or milk in the initial fluids. Gastric dilation and vomiting certainly preclude such a program. However, where tolerated it seems to be a welcome aid.

The problem of steroid replacement early in massive burns is not new. No conclusive evidence has been presented[11] demonstrating its benefit. No patient in this series demonstrated definite clinical signs of hypoadrenalism in the first 24 hr. following burn. However, 3 patients expired in an adrenal crisis following a long hospital course of multiple debridement and skin grafting. Of the total 14 burn cases autopsied, 64 per cent had definite signs of significant adrenal exhaustion, in the opinion of the examining pathologist (table I). Although cortisone therapy may mask a clinically unsuspected infection, it is equally disheartening to carry an acutely burned patient through a long clinical recovery to expire from a minor surgical procedure. In long range therapy these steroids seem indicated. It would also seem most wise periodically to evaluate the adrenal status of the burn patient by routine screening methods during the convalescent period.

We have stated that the 30 to 40 per cent group of burn patients benefit by advancing colloid and total initial fluid replacement beyond that normally given by Evans'[5] formula. In the small group of patients with greater than 50 per cent burns no such obvious correlation existed, and unfortunately, no specific conclusions can be drawn.

CONCLUSIONS

1. Evans' type formula in 30 to 40 per cent body burns does not provide sufficient initial colloid or volume of fluid replacement.

2. Correction of depleted colloid with adequate intake helps avoid prolonged anuria, and assists in alkalinization of the urine, thereby decreasing tubular hemoglobin deposition.

3. Based on clinical result and impressive autopsy findings, adrenocortical derivatives are recommended in long term management of burn patients.

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A BYPASS HOMOGRAFT FROM THORACIC AORTA TO FEMORAL ARTERIES FOR OCCLUSIVE VASCULAR DISEASE

Case Report[a]

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In this case report we describe an extension of the bypass grafting technique which has been recorded previously. The patient presented the problem of symptomatic arterial obstruction extending distally from the aorta immediately below the renal branches to the common femoral arteries. The technical problems of management were compounded by a previous unsuccessful aortic bifurcation graft done elsewhere. We placed a bypass arterial homograft from the lower thoracic aorta to the common femoral arteries and found this to be a satisfactory means for restoration of peripheral circulation.

The details of this case and a 3-year follow-up report are included below.

CASE REPORT

C. K., (KCH #342425), a 47-year-old white married ranch hand and construction worker, had been in excellent health all of his life until early in 1954 when he noted gradual onset of pain in his legs when walking. These symptoms of pain were progressive, gradually becoming more severe in the thighs. In August 1955 he noted coldness in both feet and a "blackening" of the third toe of the left foot. He was examined by his physician the following month who found absence of pulsation in the dorsalis pedis, posterior tibial and popliteal arteries bilaterally. There were no pulsations felt in the femoral artery on the right and only very weak pulsations on the left.

Angiographic studies were performed 4 days later which included an aortogram and bilateral femoral angiograms. The femoral angiograms revealed no evidence of distal obstruction in the femoral arteries although there was a suggestion of narrowing of the lumen bilaterally. The aortogram disclosed the radiopaque material (50 per cent Urokon) passing directly into the superior mesenteric artery. The aorta, bifurcation and iliac vessels were poorly outlined.

After this study, the patient's course was complicated by severe ileus, which gradually subsided over a period of 10 days at which time he was discharged from the hospital. He continued to have intermittent abdominal cramps and distension while at home.

First operation. On October 24, 1955, complete obstruction of the small bowel occurred and he was readmitted to the hospital for laparotomy. At operation, the small bowel was markedly distended down to midjejunum, at which point there was a severe constricting scar almost completely occluding the lumen. About 6 in. distally, a second zone of constriction was found. A third area of scarring and constriction was located in the proximal ileum. The remaining small bowel, as well as the colon, appeared normal. In the mesentery of the small bowel beneath the zones of scarring were areas of induration suggestive of localized aseptic necrosis. Palpation of the aorta indicated a complete obstruction at the bifurcation.

The involved areas of small bowel were isolated and resected. Continuity was re-established by end-to-end anastomosis. A total of 18 in. of small bowel were removed. Microscopically, the areas appeared as inflammation compatible with regional enteritis. The association of the onset of symptoms with the time of the aortogram was significant. Mr. K.'s convalescence following this operation was uneventful. After his discharge from the hospital he continued to gain weight.

In January 1956 he was referred to a second physician for evaluation and treatment of his vascular problem.

Second operation. On January 6, 1956, he was operated upon by the second physician and a frozen dried, ethylene oxide-sterilized homograft was implanted into the abdominal aorta. This

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graft was placed end-to-end to the aorta approximately 1 in. distal to the renal arteries and end-to-side on the left to the external iliac artery and on the right to the common iliac artery. Difficulty was encountered at the operation. Thrombosis in the graft necessitated removal of the thrombus from the graft on 2 occasions. At completion of the operation it was thought that the graft was patent and distal pulsations were palpable. Early in the postoperative period the distal pulses disappeared and 2 weeks later the patient was discharged from the hospital, his preoperative condition unimproved. His exercise tolerance was 1 block of level ground walking or 1 flight of stairs. Subsequently, the patient was referred to the King County Hospital for further care.

When examined at this hospital, no aortic pulsations could be felt. There were no pulsations palpable in the lower extremities. Oscillometric studies revealed no significant fluctuations in either leg. There was hair on the thighs and calves with soft supple skin and no evidence of severe tissue ischemia, apart from the great and second toe of the left foot which were somewhat darker in color.

FIG. 1. Lumbar aortagram exposed 6 seconds following the injection of 40 cc. of 70 per cent Urokon. There is complete obstruction of the aorta just below the renal arteries. The large number of small vessels filled with the radiopaque material denotes increased collateral circulation.

On June 4, 1956, a translumbar aortogram was performed (fig. 1). The abdominal studies show the aorta well visualized down to and just beyond the superior mesenteric artery. The aorta appeared to be completely obstructed at the level of the renal arteries. There was no apparent obstruction in any of the vessels of the thighs which were outlined by collateral blood flow (fig. 2).

Third operation. On June 7, 1956, the patient was operated upon (by L. R. S.) and the following findings were noted: The common femoral arteries bilaterally were of good caliber, soft and pliable, and without any evidence of arteriosclerotic changes. There was complete occlusion of the aorta from the renal arteries distally. The entire previous graft was embedded in dense scar tissue. The left hypogastric artery was occluded. The walls of the hypogastric artery on the right were thickened, but the lumen was patent. The upper anastomosis of the previous graft was 1 in. distal to the renal arteries. The anastomosis on the left to the external iliac artery was visualized. The anastomosis on the right, presumably to the common iliac, was not visible. The graft was surrounded by dense scar tissue, was markedly contracted and contained an organized thrombus which was not particularly adherent.

Because of the proximity of the obstruction to the renal vessels, it was felt that the best chance of aiding this patient lay in performing a bypass graft from the lower thoracic aorta down to the common femoral arteries and the right hypogastric artery.

Through a midline abdominal incision extending from the pubis to the sternum and then into the left chest through the fourth intercostal space, adequate exposure was provided. For the bifurcation, a 2-day-old homograft was used which had been preserved in saline with 10 per cent homologous serum. Added to this were 1 gm. of Chloromycetin, 1 gm. of streptomycin and 10 million units of penicillin. This graft had been refrigerated at 4°C. since its sterile removal from a 46-year-old woman 2 days previously. This graft was anastomosed end-to-side to the common femoral arteries bilaterally and end-to-end to the hypogastric artery on the right. For the upper portion, a 5-week-old ethylene oxide sterilized frozen dried thoracic aorta graft was used which had been taken from a 33-year-old donor. This was sutured end-to-side to the thoracic aorta on the left, 4 cm. above the diaphragm. The graft was brought through the diaphragm through a separate opening to the left of the esophageal hiatus and carried behind the stomach to the left of the root of the mesentery. There was an undesirable atheromatous plaque in the anterior wall of the

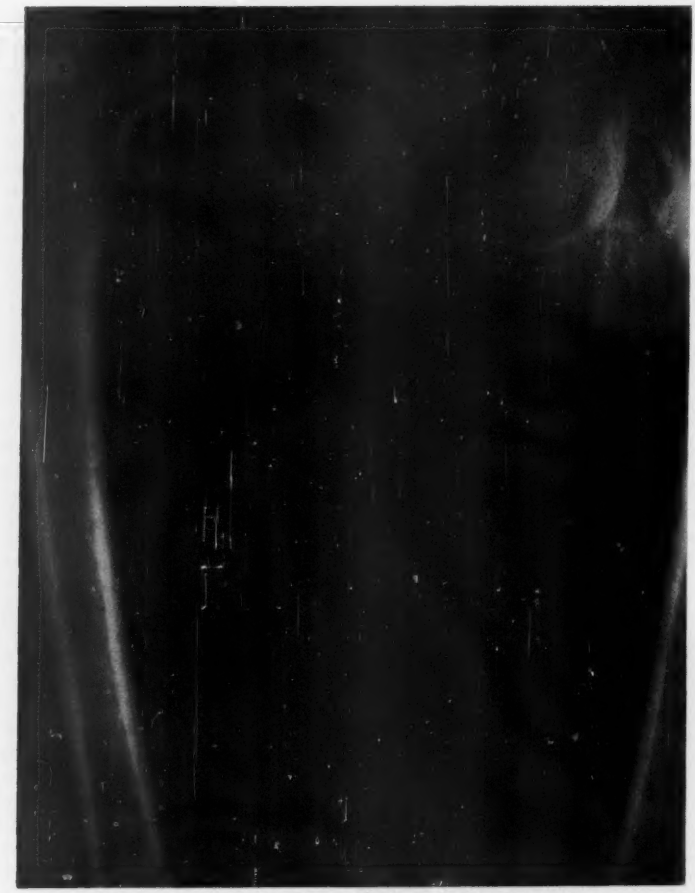

FIG. 2. Femoral arteriograms performed at the same time injecting the radiopaque material through the same needles as seen in figure 1, demonstrate some collateral filling of the arteries of the thigh with moderate caliber 12 seconds after injection of 40 cc. 70 per cent Urokon.

fresh graft located proximal to the bifurcation which was excised. The upper portion of the fresh bifurcation graft which was then free was used to connect the upper and lower graft segments. The entire graft lay more to the left of the original aorta. This resulted in having the iliac artery segment on the left too long and on the right, too short. It was remedied by excising a 4-cm. segment from the left and transplanting it into the right. In all, 9 anastomoses were performed using a continuous over-and-over 5/0 vascular silk (fig. 3).

Postoperative course. The patient's course subsequent to this extensive surgical procedure has been most enlightening. He returned to his home community to be followed by his first physician. By July 1956 he had returned to full manual labor on his own ranch. A complete examination by his doctor over a year later (October 1957) revealed normal skin temperatures from the groin to the toes and the skin color was within normal limits. Femoral, popliteal, dorsalis pedis and posterior tibial artery pulsations were strong and equal bilaterally. Oscillometric tests conducted gave 4 point fluxations in the calf and thigh bilaterally using a pressure setting of 100 mm. mercury (table 1). Summarizing the case, his doctor stated that the patient was a completely rehabilitated, happy, healthy individual and complained only of an occasional backache which occurred when he carried too many buckets of coal into the apartment house that he and his wife managed during the winter months (fig. 4).

The patient continued to do well until January 1958 when he first noticed increased dyspnea and occasional "black out" spells following exertion. On February 15, 1958, while out walking, he experienced sudden onset of severe claudication in the right calf. Upon examination at that time, there was complete absence of pulses in the right leg which was noticeably cooler than the left. In the left leg there were good femoral artery pulsations, weak popliteal and posterior tibial and absent dorsalis pedis artery pulsations. Good skin sensation and normal active reflexes were present bilaterally. The patient was referred to the King County Hospital on February 17, 1958, and the above findings were confirmed. An attempt to perform an aortogram through the right brachial artery was unsuccessful because of severe atheromatous changes in the vessel wall which made it impossible to advance the small catheter beyond the right subclavian artery.

On the evening of March 7, 1958, while ambulatory in the hospital, the patient experienced sudden severe claudication in the left foot and ankle. On examination, there was stocking hyperesthesia from midcalf distally. Femoral and popliteal pulses on the left were present but diminished. All pulses of the right leg were persistently absent. A continuous epidural block was started and anticoagulant therapy was instituted. Under constant observation during the night, the left popliteal and femoral artery pulsations disappeared. The following morning, March 8, 1958, the left femoral and popliteal arteries were explored surgically under local anesthesia. A fresh thrombus was easily evacuated from the previous homograft vessel, which resulted in excellent blood flow. There was severe atheromatous degeneration of the host femoral vessel distal to the graft. This was also true of the popliteal artery which was completely occluded distally by these changes. On March 18, 1958, the patient gave permission for left hip disarticulation which was necessary in order to remove all of the nonviable tissue. After extensive physiotherapy and crutch walking exercise, the patient was discharged from the hospital on May 30, 1958. Severe generalized atheromatous changes contraindicated any surgical procedures to the remaining leg.

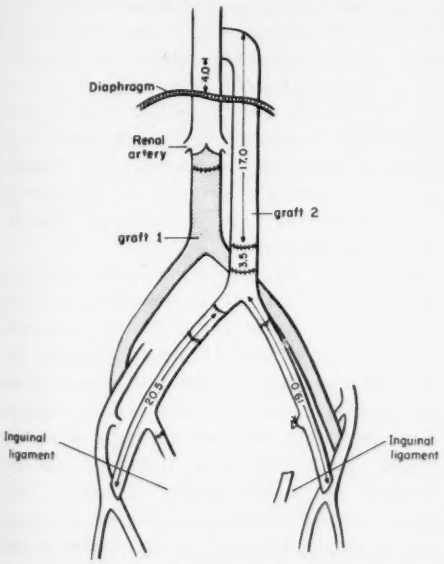

FIG. 3. Diagramatic illustration of the bypass homograft completed. The *stippled area* represents the previous unsuccessful homograft with thrombus extending upward to the level of the renal arteries.

TABLE 1

Oscillometric study (cuff set at 100 mm. Hg)

Location	Fluctuations and Date			
	*Preoperative** 6/3/56	*Postoperative** 7/4/56	*Following hemograft bypass* 10/7/57	*Last admission* 2/19/58
Right arm....	3	3		
Left arm.....	3	3		
Right thigh...	0	4	0.5	4
Left thigh....	0	2.5	3	4
Right calf.....	0	3.5	0.2	4
Left calf.....	0	2.5	3	4

* Date of operation, 6/7/56.

Pathologic examination of the amputated left leg showed severe atheromatous degeneration of the femoral, profundus femoral, popliteal and posterior tibial arteries with complete obliteration of the lumen in localized areas. There were old organized and liquified thrombi present in all the major collateral vessels of the thigh and leg. On examination in November 1958, the patient was moderately active. However, he was unable to walk more than 75 yards on crutches without tiring. Examination of the pulses at that time was as follows: upper extremity, brachial absent bilaterally; right ulnar and radial 2 +; left ulnar and radial 1 +; lower extremity right, femoral artery just detectable, popliteal absent, dorsalis pedis, 1 +; and posterior tibial 1 +.

The patient remained moderately active, performing sedentary jobs about the apartment house until February 1959, when he was hospitalized for 3 days for mild left hemiplegia which improved with symptomatic treatment. He was

FIG. 4. Photograph of the patient ambulatory on the ward on the 19th day following the homograft bypass procedure. The extent of the thoracoabdominal incision with secondary incisions can be seen well healed.

readmitted to the hospital in August with complete paralysis of the left side of the body, right facial paralysis and aphasia. There was progressive increase in paralysis leading to his death on August 23, 1959.

Autopsy examination revealed severe generalized atherosclerotic changes of all major arteries. The abdominal aorta was completely occluded from the renal arteries distally. The arterial homograft extending from the thoracic aorta just above the diaphragm to the iliac vessels was completely occluded at the ostium by old organized thrombus which extended the entire length of the graft.

DISCUSSION

The procedure detailed in this report is an extension of the bypass end-to-side graft technique introduced in France by Kunlin[2] and popularized in this country by Linton and Menendez[3] and Crawford and De Bakey.[1] Further observations will be necessary to ascertain the full value of this extension of the bypass graft technique. However, the results of a 38-month follow-up in this particular case indicate that under some circumstances it may have some merit.

One of the most interesting aspects of this particular case history is the fact that the homograft remained patent and functioning for such a long period of time in the face of a continuing progressive generalized atheromatous change in the host vessels. Although it is true that the homograft ultimately became occluded by thrombus, one can assume that this was due to decreased flow and turbulence secondary to the gradual but progressive decrease of an effective distal vascular bed. Snyder and Campbell[4] have shown experimentally that atheromatous change in the aorta of rabbits is impeded distal to a ligature partially occluding the aorta.

They feel that this marked change is secondary to a drop in pressure distal to the partial occlusion. This experimental situation is analogous to the patient presented here with occlusion of the distal aorta and bifurcation with a good patent distal vascular bed. Thus the question is raised—if we operated and remove this obstruction, do we permit or actually hasten atheromatous degeneration in a previously patent, apparently nondiseased distal bed?

We believe that this case history illustrates again that we actually do very little to alter favorably the course of this disease process by operative procedures. It also emphasizes the need for a combined approach to these difficult problems. Modifications of diet, plus the addition of a medication that will not only effectively "block" the absorption of cholesterol from the intestine but safely control cholesterol biosynthesis in the tissues as well, is necessary in the hope of altering the course of this disease process favorably. If this is not done, we then limit ourselves to alleviating or perhaps just delaying some of the symptoms of the disease that progresses to the ultimate death of the patient.

Acknowledgment. We are greatly indebted for the continued cooperation and follow-up studies of the latest case reports from Dr. Ausey H. Robnett, Spokane, Washington.

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INTRA-ARTICULAR HEMANGIOMA OF THE KNEE

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Hemangioma, a common benign vascular tumor originating from endothelial tissue, can arise in all parts of the body, with the exception of cornea, cartilage, hair and nails. Approximately one-half of hemangiomas occurring in the lower limb are located within or about the knee joint. Intra-articular hemangioma of the knee, first described in 1856 by Bouchut,[1] is a rare lesion, there being less than 60 such cases reported in the world literature to date.

CASE REPORT

A 17-year-old Caucasian male presented with limping and pain, behind and just above the left patella, of 4½ months' duration. The pain was dull and continuous, being intensified by locomotion. The patient had palpated a retropatellar, vaguely delineated, soft mass approximately 3 months before being seen. Two months prior to hospitalization he had been struck on the left patella with a baseball, which injury had resulted in increase of the dimensions of the mass and augmented his incapacitation. For the past 4 months he had been treated elsewhere by diathermy, massage, muscle relaxants, analgesics and restricted weight-bearing on the affected extremity, without relief of his symptoms.

The system review and family history were noncontributory.

Physical examination revealed a youth in obvious distress. Significant findings were limited to the left lower limb. The latter showed no length discrepancy, and muscle atrophy was absent. There were no cutaneous vascular disorders. The knee joint was moderately tender throughout. Effusion, crepitus or signs of internal derangement were nonexistent. Motion of the joint was limited to approximately 20 degrees in flexion, with pain being elicited by manipulation. A soft, compressible mass, estimated at 3 cm. in diameter, could be barely outlined behind the patella. A thrill or bruit was not evident, and peripheral pulses were full and comparable to those of the contralateral extremity.

A plain roentgenogram of the knee was unremarkable. Pneumarthrography was not performed.

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The initial impression was peri- or intracapsular cyst or lipoma, and in view of the patient's failure to respond to conservative management, he was subjected to exploratory arthrotomy.

An elliptical incision, approximately 4 cm. in length, was made at the upper border of the patella and extended medially to just below the horizontal joint line. The tendon of the rectus femoris was transected and reflected, the knee flexed, and the joint penetrated. A tumor mass immediately came into view. It was of a dark reddish-brown color, like fresh chicken liver, and was adherent intimately to the entire lateral portion of the synovial membrane and to some of its medial aspect also. The tumor was sessile, papilliferous and very vascular. Its gross appearance was not much unlike that of a villonodular synovioma. The entire synovial membrane and mass were excised, including a section of the underlying capsule, the better to control hemostasis (fig. 1). The wound was closed in layers, the limb enveloped with an elastic bandage and extended on a plaster of Paris splint. One week after the operation the patient was started on physiotherapy and graded ambulation. His convalescence was uncomplicated, and he was discharged 3 weeks postoperatively. He has remained asymptomatic at the time of this writing, 6 months later.

The histologic sections disclosed prominent collections of thin-walled blood vessels with large lumina. The synovial tissue showed nonspecific chronic inflammation with a moderately dense infiltration of chronic inflammatory cells. A minimal amount of hemosiderin was present (fig. 2). The pathologic diagnosis was cavernous hemangioma of the knee joint with chronic nonspecific synovitis.

DISCUSSION

Clinical features. Because an articular angioma is a lesion rarely encountered, the ease of error in diagnosis is readily admitted. Contrariwise, a number of factors make it possible to suspect this tumor, especially since it is rarely found elsewhere but the knee joint.

A history of hemangioma in the patient or members of the family is significant. The age incidence varies from 3 to 37 years; most subjects,

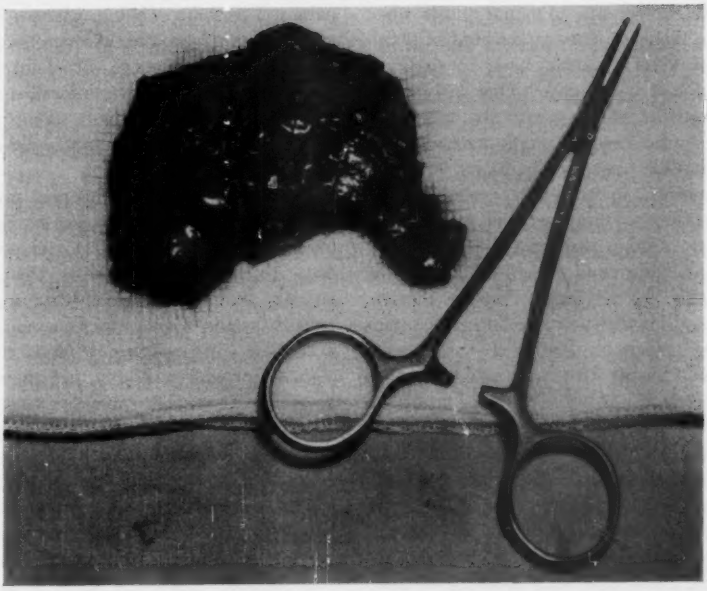

FIG. 1. Excised specimen, which had a maximum diameter of 6.2 cm and a thickness of approximately 3.4 cm. The upper two-thirds represents tumor tissue. The papilliferous projections are collapsed in the photograph.

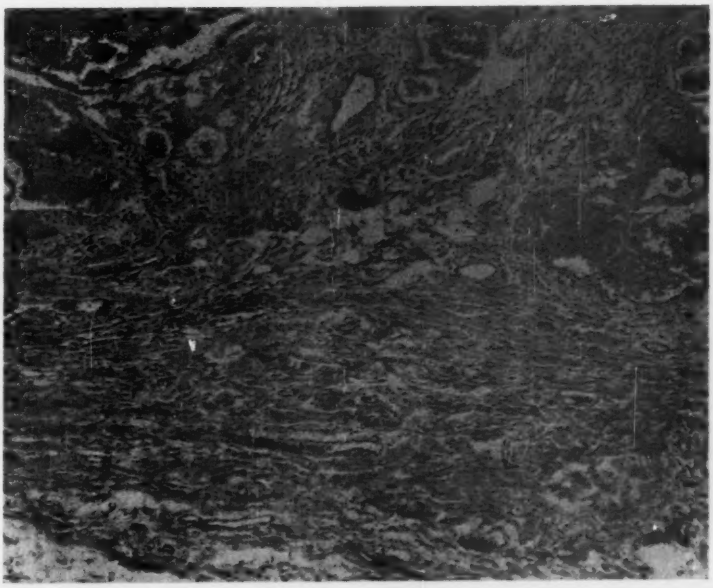

FIG. 2. Histologic section, revealing blood filled sinuses, hemosiderin, dense connective tissue and chronic inflammatory cells.

however, are adolescents or young adults when first seen. In many of these an account of manifestations of disease dating back to early or middle childhood is obtained. This early onset of symptoms is in harmony with the concept of a local congenital vascular malformation being the most plausible basis for this lesion. Antecedent injury, when in evidence, probably only directs attention to the condition rather than having direct causal relationship.

The clinical features of articular hemangioma are nonspecific, in the main. Pain on motion, tenderness and intermittent swelling of the joint occur almost uniformly. The pain is augmented by elevation of the leg. Other more common signs are a limp, slight or moderate limitation on extremes of motion, visible atrophy of the quadriceps, and palpation of a doughy, elastic mass which tends to diminish in size if the extremity is raised. Less frequently there may be leg length discrepancy, crepitus and signs of internal derangement of the joint. In approximately one-fourth of the cases cutaneous mottling or associated soft tissue hemangiomata, locally or elsewhere, are present and of diagnostic significance.

Puncture and aspiration of the joint may release variable amounts of sanguineous fluid if the needle is fortuitously plunged directly into the tumor; otherwise the yield will be normal synovial fluid. Plain, stereo-, or pneumoroentgenography is usually unreliable, or it may disclose a vague soft tissue shadow in the region of the affected joint. The presence of phleboliths in this soft tissue mass is helpful as representing organization and calcification of thrombi in the lesional vascular tissue. The phleboliths have the appearance of round, lamellated, dense bodies varying in size from that of a head of a pin to that of a pea. Arteriography should probably be considered more often as a diagnostic adjunct.

The differential diagnosis includes: lymphangioma of the capsule, chronic infectious granuloma, pigmented villonodular synovitis, bursitis, rheumatoid arthritis, internal derangement of the joint, lipoma of the infrapatellar fat pad and other benign tumors.

Pathologic findings. Bennett and Cobey[2] have divided synovial hemangiomas into two categories according to the extent of involvement—circumscribed or localized and diffuse or infiltrating. The circumscribed lesions may be stalked or sessile. The diffuse variety more often cause only pain and periodic swelling, whereas the localized type ordinarily produce characteristic symptoms arising from interference with joint function.

The tumor is grossly vermiform, is covered by a thin bluish capsule, and has a spongy consistency, revealing on cut section innumerable interwoven vascular sinuses.

Microscopically, hemangiomas fall into four classes, as described by Stout:[3] capillary, cavernous, mixed capillary and cavernous, and venous. In the capillary forms there are intertwining, endothelial, blood-filled spaces in haphazard fashion. Cavernous hemangiomas present a similar picture except that the vascular sinuses are widely dilated and show much proliferation of compact connective tissue. In venous hemangiomas the vessels have relatively thick walls and contain smooth muscle cells. The connective tissue may be studded with considerable refracted brown pigment. Inflammatory cellular components, which may or may not be lacking, bear no correlation to clinical findings.

Treatment. Since the knee is an important weight-bearing joint subject to irreversible arthritic changes in long standing disease processes, early arthrotomy, even in the presence of a doubtful diagnosis, should be considered in every instance where articular hemangioma is suspected. When the tumor lends itself to total excision the relief of symptoms and restoration of joint function obtained thereby are striking. Only too frequently patients with this condition are placed on a so-called "watchful expectation" regimen of analgesics, tranquilizers, diathermy, exercise, encasement of their limbs in plaster of Paris and steroid infiltrations of their joints, with little or no relief from their disability.

In nearly all the cases documented surgical excision seems to have been the method of choice. The ideal lesion to treat is the discrete, pedunculated type which is removed with a wide berth. The likelihood of recurrence is practically nil in such instances. If the case is one of diffuse involvement, the treatment is synovectomy and wide resection of adjacent soft tissues, with or without postoperative irradiation. The difficulty of eradicating the lesional tissue completely in this variety favors recurrence. Involvement of the regional muscles or bones, in addition to the articular contents, frequently results in incomplete excision of the tumor, extensive hemorrhage, and increased likelihood of recrudescence.

Bennett and Cobey[2] advise either roentgen ray or radium therapy in these situations. One of their patients, an 8-year-old girl, required amputation.

Prognosis. The ultimate outcome depends on the extent and duration of the tumor before treatment, and on the feasibility for its complete excision, rather than on any inherent pathologic characteristics.[4] Recovery may be accompanied, however, by some limitation of joint motion. Recurrence is the almost invariable penalty for incomplete excision of the lesion. A good prognosis is jeopardized by a long standing, diffuse hemangioma in the soft tissue affecting the bone and joint, resulting in shortening or lengthening of the extremity, weakened bones with possibility of pathologic fractures, atrophy of muscles and changes in the surface of the joint.

Finally, it is worth noting in connection with the prognosis that there appears to be no case on record in which a capsular hemangioma has undergone spontaneous malignant transformation.

SUMMARY

Intra-articular hemangioma is an uncommon tumor of childhood or adolescence which affects the knee almost exclusively, and it is seldom diagnosed preoperatively. It is characterized by a variety of nonspecific symptoms and signs, the cardinal ones being pain, the presence of a mass, and intermittent swelling and limitation of motion of the joint.

Wide excision of the tumor, with or without postoperative irradiation, is the indicated treatment. The pedunculated, circumscribed type of tumor, being more amenable to complete removal, offers the best prognosis. The infiltrating variety, which sometimes leads into periarticular soft tissues, has a less favorable prognosis.

A case is described of diffuse intra-articular cavernous hemangioma of the knee which was successfully excised, with complete resolution of symptoms and maximal return of joint function.

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THE TREATMENT OF WOUNDS WITH 3-AMINOPHTHALHYDRAZIDE

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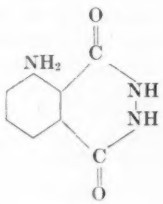
3-Aminophthalhydrazide was first synthesized by Schmitz[9] in 1902. Its chemical structure is as follows:



This substance is only slightly soluble in water and organic solvents, but freely soluble in acid and alkali. In neutral and weak acid solution, it reveals very strong fluorescence; it emits strong luminescence when an oxidizing agent is added to it in the alkaline solution. In 1928, this phenomenon was studied by Albrecht[1] in Germany with careful observation and consideration from the standpoint of physical chemistry, and the reactive process was postulated as shown in scheme 1.

(I) is the molecular form of the above substance either in the neutral or acid solution and (II) is that in the alkaline solution. When this substance is oxidized by oxidizing agents, such as hydrogen peroxide, ozone, hypochlorite, and so forth in the alkaline solution, it becomes an oxide of the form (III), which is partially hydrolyzed to aminophthalic acid (IV) and diimide. The latter, diimide, being unstable and strong in reduction power, acts on (III) reducing it to the first molecular form (I) and the luminescence arises.

Gleu and Pfannstiel[2] later found that hemin increased luminescence, acting as a catalyst in the reaction described above.

It is suggested that 3-aminophthalhydrazide with α,β-unsaturated ketones may exhibit wide general biologic activity as an oxidation-reduction catalyst. In view of this activity, some biologic responses of 3-aminophthalhydrazide[3-8] were observed. With these theoretic considerations in mind, a further clinical trial on wound healing using 3-aminophthalhydrazide made up as a solution and ointment was carried out.

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MATERIALS AND METHODS

A 0.05 per cent 3-aminophthalhydrazide in sterile physiologic solution with a pH of 7.2 was prepared. Gauze dressings were soaked with the solution and applied totally to purulent wounds once a day.

For nonpurulent and granulation wounds, an ointment composed of 0.1 per cent 3-aminophthalhydrazide in petrolatum base was used.

In order properly to evaluate 3-aminophthalhydrazide therapeutically, scarlet red in petrolatum base was used as the control in the treatment of nonpurulent wounds and burns. Irregularly contoured wounds were avoided for this study. A wound was divided into two parts by a marking label, or patients with two or more similar wounds were selected. 3-Aminophthalhydrazide ointment was applied on a counterpart and scarlet red ointment on the other. Observation of wounds was made every day.

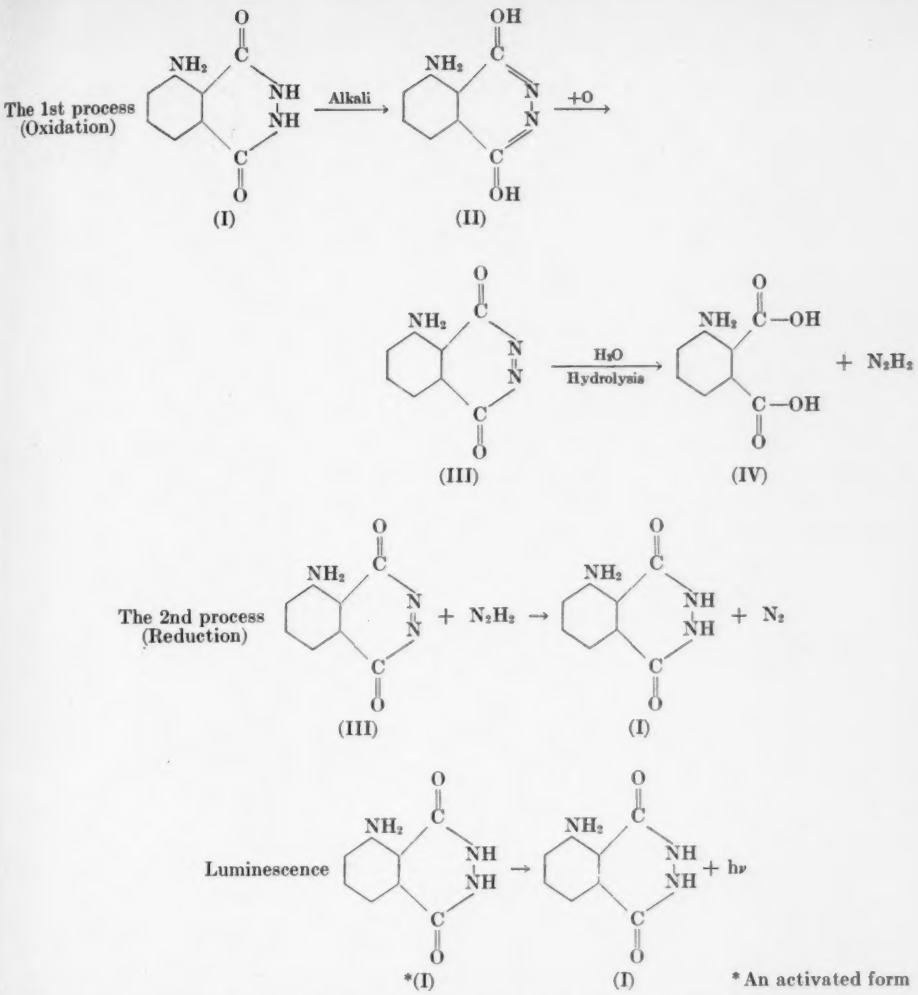
RESULTS

The results of wound healing treated with 0.05 per cent 3-aminophthalhydrazide solution are summarized in table 1. In 30 cases of nonpurulent wounds, 22 (73 per cent) showed good response; 1 (3 per cent) revealed poor healing. Of 35 cases in purulent wound, 13 (37 per cent) exhibited good response; 7 (20 per cent) were poor.

As shown in table 2 all the nonpurulent wounds treated with 3-aminophthalhydrazide ointment healed more rapidly than with scarlet red by an average of 3 days.

CASE REPORTS

Case 5. Postappendectomy incision of a 25-year-old woman with a secondary healing wound 16 by 5 cm., was divided into two parts and followed by the comparative method. After 3 days 3-aminophthalhydrazide ointment side showed



SCHEME 1. Hypothetical process of the chemiluminescent reaction of 3-aminophthalhydrazide

diminution of serous secretion, with good proliferation of fibroblasts; on the other hand the scarlet red side showed atonic granulation without diminution of secretion. After 1 week, epithelization was proceeding on the 3-aminophthalhydrazide side and the size of wound was slightly reduced. After 3 weeks the 3-aminophthalhydrazide side revealed completion of epithelization and closure of wound. The scarlet red side did not completely epithelize until 32 days.

Case 13. Burns on the right leg (third degree) of a 36-year-old man. Two of several burns 3.0 by 2.4 cm. and 3.6 by 2.6 cm. were observed comparatively. One was treated with 3-aminophthalhydrazide ointment, the latter with scarlet red ointment. After 3 days the 3-aminophthalhydrazide side showed a decrease of secretion and proliferation of crimson granulation; the scarlet red side showed much secretion and pinkish granulation. After one week the size of wound was 2.4 by 1.7 cm. on the 3-aminophthalhydrazide side, and 2.8 by 2.1 cm. on the scarlet red side without secretion; after 2 weeks it was 1.0 by 0.8 cm. in the former and 1.5 by 1.2 cm. in the latter. After 3 weeks the 3-aminophthalhydrazide side produced complete epithelization. The scarlet

TABLE 1

Response to 3-aminophthalhydrazide therapy in wound healing

Wound	Method of Use	Total No. Cases	Good*	Fair†	Poor‡
Acute purulent	Solution	15	5	8	2
Chronic purulent	Solution	20	8	7	5
Pyrogenic	Solution	13	6	4	3
Tuberculous	Solution	7	2	3	2
Nonpurulent	Ointment	20	14	6	0
Granulation	Ointment	10	8	1	1
Secondary healing	Ointment	4	4	0	0
Burns	Ointment	6	4	1	1

* Good: marked accelerated healing.
† Fair: slight accelerated healing.
‡ Poor: delayed healing.

TABLE 2

Comparative results of nonpurulent wound healing between 3-aminophthalhydrazide and scarlet red

Case No.	Therapy Used	
	3-Aminophthalhydrazide	Scarlet red
	healing time days	
1	13	16
2	10	11
3	12	15
4	16	20
5	21	32
6	18	24
7	13	18
8	6	6
9	12	15
10	19	24
11	8	10
12	13	15
13	21	25
14	5	5
15	8	10
16	12	15
17	15	19
18	9	11
19	18	22
20	12	15
Average	13	16

red side was 0.8 by 0.4 cm., and it finished epithelization after 25 days.

DISCUSSION

It should be noted that in this study wound healing treated with 3-aminophthalhydrazide was accelerated appreciably in 88 per cent of wounds observed. The lower percentage of poor healing in purulent wounds, as compared with the percentage in nonpurulent wounds, suggests that 3-aminophthalhydrazide is not strictly bactericidal.[3–5] It does exert a definite bacteriostatic effect *in vitro*, by reason of its interference with oxidation-reduction mechanism. The precise mode of this interference had not been determined.

Clinical investigation has demonstrated the successful use of 3-aminophthalhydrazide as a local agent in the surgical treatment of certain acute and chronic infections.

SUMMARY

1. 3-Aminophthalhydrazide has been found to be an effective agent in the local treatment of acute and chronic infected wounds as well as noninfected wounds and burns.

2. 3-Aminophthalhydrazide was highly effective in stimulating the proliferation of fibroblasts and epithelization in wounds and burns.

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LYMPHOSARCOMA OF THE SMALL BOWEL AND COLON: REVIEW AND REPORT ON ONE CASE OF EACH

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Lymphosarcoma of the intestinal tract is an idiopathic change of the lymphoid tissue and lymph nodes in which the stem cells have differentiated along the lymphocytic line.[6] It is characterized by arising in a group of lymph nodes or tissue in the submucosa of the bowel. From this site it may spread to adjacent tissues by infiltration or by way of the lymphatics. Further and later involvement may occur in distant tissue by: (1) true metastasis by way of the lymphatics or blood stream; or (2) malignant changes in the lymphoid tissue at other sites as an expression of the same malignant potential as that at the original site.[3, 8, 29]

The uncommonness of lymphosarcoma of the intestinal tract has prompted a number of papers. Libman[20] in 1900 added 5 cases of lymphosarcoma of the intestinal tract to those previously reported by Baltzer in 1893. He noted that: (1) in the period from 1859 to 1875 the Berlin Pathological Institute had recorded no cases; (2) Smolner found 13 cases of lymphosarcoma of the bowel in 13,036 autopsies done in Prague over a 15-year period; and (3) Nothnagel in 1905[27] had reported 9 cases of lymphosarcoma of the small bowel over a 12-year period during which time 21,359 autopsies had been accomplished and reviewed. During this time there were 274 cases of sarcoma of all types and 243 carcinomas of the small bowel. By 1926 Minot and Isaacs[25] had accumulated 477 cases of lymphosarcoma of the bowel. They also pointed out that the incidence was higher in men than women, 5 to 2. The duration of symptoms was about the same in both sexes. Lymphosarcoma of the small bowel compared with the large bowel was 2 to 1 as reported by Ullman and Abeshouse.[42] In Raiford's[33] study of 88 tumors of the small bowel, both benign and malignant, there were 21 lymphoblastomas. A review of 157,488 admissions at the Montreal General Hospital for the period 1905 to 1930 revealed only 6 cases of lymphosarcoma of the intestine.[1] During a 25-year period at Jewish Hospital, Brooklyn, 15 cases of lymphosarcoma of the large and small bowel were found. In the same time period 48 carcinomas of the small intestine, 1377 carcinomas of the colon and 577 carcinomas of the rectum were identified.[14, 31] By 1955 Stout[38] was able to review 238 cases of noncarcinomas and nonadenomas of the colon and rectum; there were 36 lymphosarcomas, 13 of the colon, and 23 of the rectum.

The clinical manifestations of small bowel involvement are not typical of lymphosarcoma alone. A correct preoperative diagnosis is rare.[22, 38] Most frequently mentioned are mechanical obstructive symptoms such as cramping with occasional vomiting and melena.[2-4, 9, 12, 17] Usually weight loss follows quickly and an abdominal mass may be noted in the disease. The new growth may produce symptoms by encircling the bowel and closing the lumen, or be the cause of an intussusception. On examination the findings depend upon the level of obstruction, the rapidity of the development of the obstruction and the completeness of the obstruction. Often a mass may be palpated. Anemia due to blood loss can be present.[8, 40] Occasionally the presenting difficulty is perforation and resulting peritonitis.[28, 30] Roentgen film studies are valuable in determining the site of the pathologic condition,[7, 11] but are not typical or characteristic of lymphosarcoma alone.

The treatment of choice is surgical removal of the local lesion, if possible, followed by radiation to the area and any regional node involvement. Supportive management is determined by the total problem presented by the patient.[2, 7, 8, 12, 17, 24, 39, 41, 42] About two thirds of the cases are not operable.[7]

Prognosis will depend on: (1) whether the local lesion can be removed completely; (2) presence of any metastasis at the time of surgery; (3) response to radiation therapy; (4) development of metastasis subsequent to surgery.[41] The immediate postoperative deaths have been lessened from the loss of 4 out of 5 patients in 1900[20] to 1 out of 8 in 1952[12] by improved anesthesia, fluid and electrolyte control

and blood volume maintenance techniques. Five-year survivals vary from none for rectal sarcoma[46] to from 10 to 30 per cent for those found in the other parts of the intestine.[7, 12, 22, 26, 35]

The incidence of intestinal lymphosarcoma in this hospital from January 1950 to January 1960 was limited to the 2 reported cases. During this period there were 45,151 admissions and 835 laparotomies performed for lesions of the large and small bowel. This included appendectomies. The total number of carcinomas for this period were 100 of the small and large bowel, and 57 of the stomach.

Since this paper was prepared, an additional case of multiple lymphosarcomas of the small bowel has been seen at this hospital with resection of the involved small bowel followed by radiation therapy. This patient is now doing well, some 4 months after surgery.

CASE REPORTS

Lymphosarcoma of the jejunum. This veteran was first seen at this hospital in December 1953 at the age of 43 for a service-connected back injury. Preceding admission he had 2 to 3 weeks of heartburn, gaseous distention, and a sense of fullness even on eating a small amount of food. There had been intermittent vomiting. He had noted an 18-lb. weight loss in the preceding three months. He was not aware of any change in bowel habits, was not constipated and did not recognize any food intolerances.

Physical examination at that time showed a moderate abdominal distention and some tympany in the left upper quadrant, hyperactive bowel sounds and some mild limitation of flexibility of the lower back.

Admitting hemogram showed a normal hemoglobin, hematocrit, leukocyte count and differential count. Kahn test and malaria smears were negative. Fasting blood glucose, urinalysis, serum bilirubin, cephalin flocculation and prothrombin times were normal. Nonprotein nitrogen was slightly elevated to 37 mg. per cent. Serum protein was 7.0 per cent with a normal albumin-globulin ratio. Serum amylase was 81. Serum agglutinations were within normal limits for typhoid, paratyphoid, brucella, proteus and tularemia.

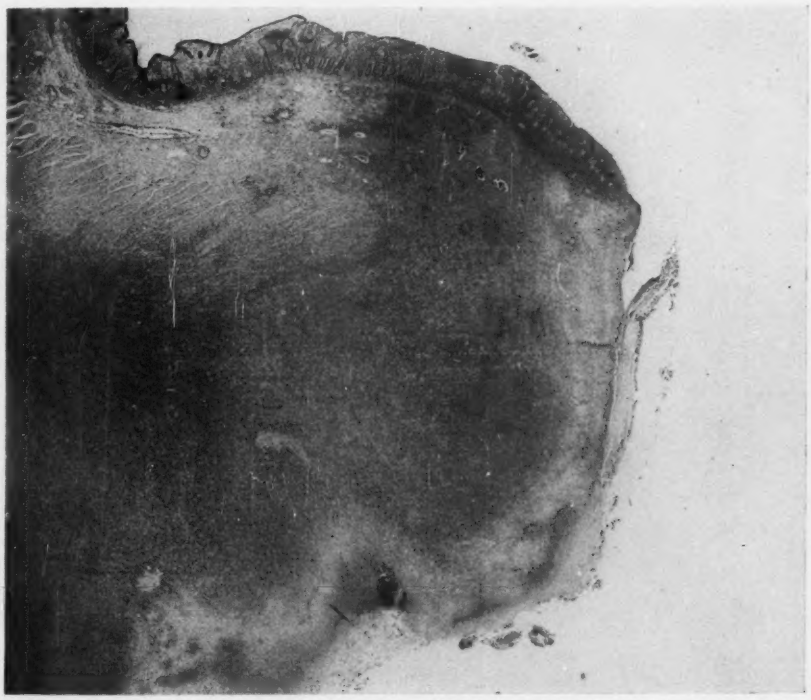

FIG. 1. Low power photomicrograph of a large ulcerating lesion of the ileum. The margin of the ulcer is thickened and rolled. Note the destruction of all layers of the wall.

X-ray of the chest, intravenous pyelogram and colon study were within normal limits. Films of the abdomen showed multiple loops of mild to moderate distention of the ileum with a few small fluid levels. Gall bladder studies were normal. Upper gastrointestinal studies were interpreted as showing mild prolapse of the prepyloric mucosa into the duodenum.

The man was discharged as undiagnosed disease of the bowel manifested by gaseous distention, abdominal cramps and heartburn.

Nearly 5 years later, in April 1959, he was admitted after 15 days of chills, fever, malaise, weight loss and some cough. There had been abdominal discomfort and diarrhea for 2 months. No melena, hematemesis and no jaundice had been noted.

Physical examination showed no significant change from the evaluation 5 years before. Laboratory evaluations again showed a normal urinalysis, serology and hemogram. Stools were negative for ova and parasites. Sputum cultures were negative. Urine culture showed a hemolytic nonpigmented forming coagulase negative micrococcus. Electrocardiogram was normal. Chest x-ray showed small infiltrates in the perivascular and perilymphatics in both lung fields. Colon study was normal. Gall bladder studies were normal. Esophagus, stomach and duodenum were considered to be normal. He had no fever during this period of hospitalization. Discharge diagnosis was possible psychophysiologic gastrointestinal reaction.

Final admission was 2 months later, in June 1959. This was for abdominal pain in the left lower quadrant for 24 hours with nausea, vomiting, intermittent stomach-ache, marked exhaustion for 4 to 5 weeks and chills and fever for a 2-week period. There had been normal bowel movements. Admitting temperature was 101°F.; pulse, 84; blood pressure, 132/80. He was an acutely ill white man who had some inspiratory wheezing in the left lower chest. Abdomen was moderately tense with diffuse tenderness. The bowel sounds were decreased and the abdomen was tympanitic. A small firm nodule was felt in the rectum in the posterior midline. Remainder of the physical findings appeared unremarkable.

The admitting leukocyte count was 8100. The following day it was 14,500, with 91 per cent polys and 8 per cent lymphocytes. Urinalysis showed a trace to 1 plus albumin. Serology was negative.

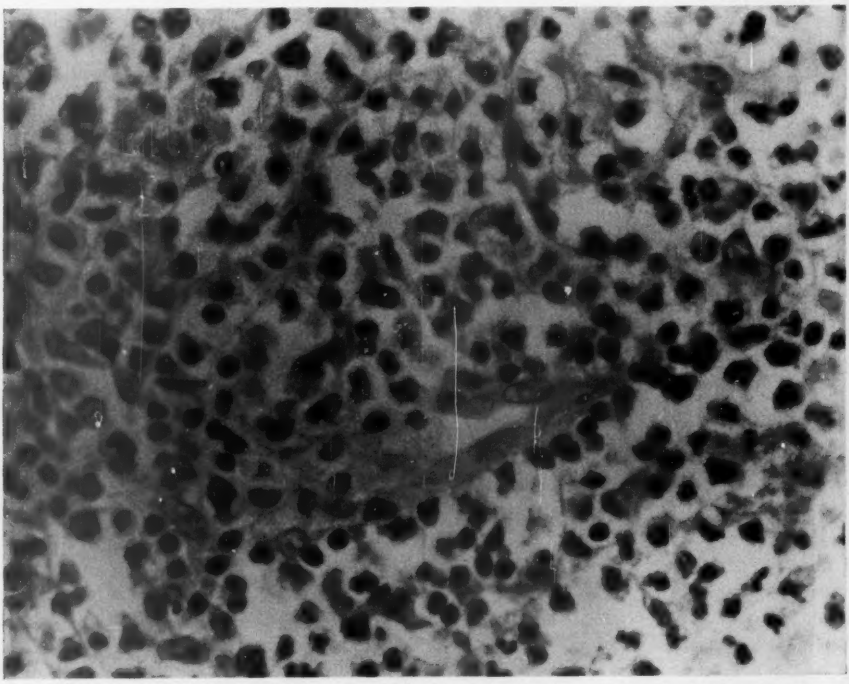

FIG. 2. High power (405 ×) photomicrograph showing sheets of small neoplastic lymphocytes with occasional large reticulum-like cells. These are numerous bizarre mitoses.

Cultures and electrolytes were not remarkable during this period.

Chest x-ray was interpreted as being within normal limits. Admitting flat plate of the abdomen showed ileus with no evidence of perforation of a hollow viscus. The following day there was air seen under the domes of the diaphragm and interpreted as perforation of a hollow viscus. The ileus was still present.

This man was operated upon the day following admission and found to have multiple ulcers of the jejunum with one perforation. Multiple jejunal resections with end-to-end anastomoses were required. The diagnosis of lymphosarcoma of the jejunum at multiple sites was established on these tissues (figs. 1 and 2).

On July 9, 1959, upper gastrointestinal study was carried out and showed segmentation of the small bowel with dilated small bowel loops. The patient made what appeared to be a satisfactory recovery until 2 months after the operation when temperature spikes and skin lesions in various areas appeared. One lesion on the left arm became ulcerated (fig. 3). It was the consensus of opinion at that time that this was due to diffuse spread or involvement by the lymphosarcoma. Mustargen was given in the amount of 10 mg. in saline on 8 successive days. He became progressively weaker, developed a leukopenia with a leukocyte count of 2350, no agranulocytosis, and a moderate anemia with hemoglobin of 10.5 gm. He continued his downhill course despite various supportive measures and died after a period of pyrexia. The highest temperature was 106°F.

Lymphosarcoma of the colon. At the age of 80 this Spanish-American War veteran was evaluated in August 1958 for prolonged constipation. In the 6 months before admission his difficulty had become progressively worse and he had required enemas and laxatives. In the preceding 3 weeks he had not been able to have any bowel movements without resorting to medication. There was cramping in both upper quadrants and considerable distress and discomfort in the few days before admission. He had noticed no blood in his bowel movement and there had been no nausea or

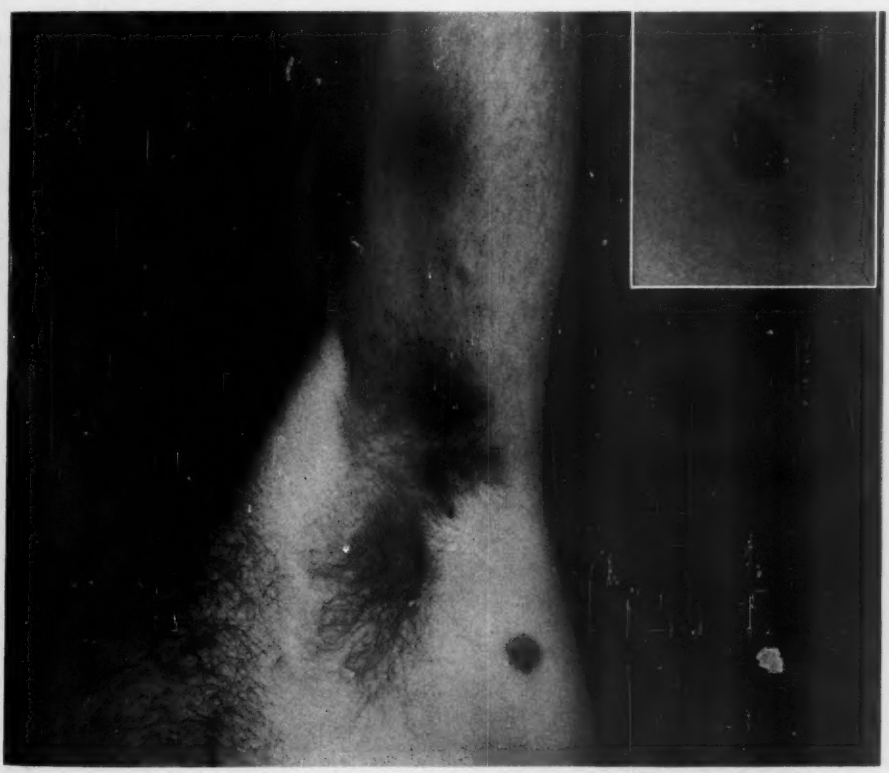

FIG. 3. Lymphosarcoma skin lesions with a close-up of the ulcerating lesion

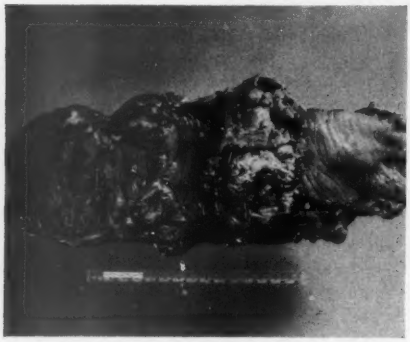

FIG. 4. Resected transverse colon showing a large fungating tumor mass having the gross characteristics of carcinoma.

vomiting. In the past there had been episodes of renal hematuria for no demonstrated cause. This man had been seen in this hospital about 5 years earlier for prostate and urinary trouble and pulmonary emphysema with occasional episodes of pulmonary embarrassment. Through subsequent periods of observation, his blood pressure had remained in the neighborhood of 120/70. His lung findings had remained the same during this time.

On this particular examination there was slight abdominal distention, but the abdomen was soft and only mildly tender to palpation. No masses were identified. In contrast to his previous normal leukocyte counts, it was now elevated to 11,600 with 73 per cent neutrophils, 20 per cent lymphocytes, 5 per cent monocytes and 2 per cent basophils. His hemoglobin and hematocrit had been good through all previous visits and was normal on this visit at a value of 12.9 gm. hemoglobin and hematocrit 43. Microscopic urinalysis on examination showed many white blood cells and red blood cells and was positive for occult blood. This finding had been present in the last 2 or 3 hospitalizations. On this particular occasion it was found that he had a hiatal hernia and obstruction in the splenic flexure portion of the colon. The x-ray impression was a carcinoma or malignancy of the colon. He was submitted first to decompression colostomy and later extirpation of the malignancy. This tissue, when studied, revealed lymphosarcoma of the left transverse colon with penetration of all layers of the bowel, but without

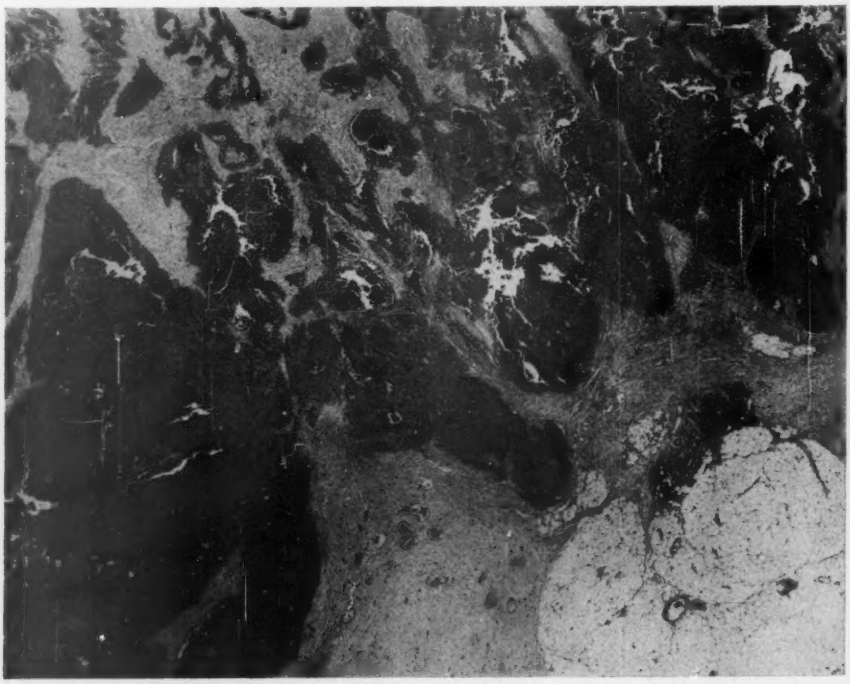

FIG. 5. Lower power photomicrograph showing sheets of invasive neoplastic cells penetrating through the muscularis externa of the colon.

definite evidence of lymph node involvement (figs. 4 and 5). At the time of discharge his hemoglobin was 12.2 gm.; sedimentation rate, 16; hematocrit, 40.

Approximately 1 year later on follow-up his blood pressure was still normal at 112/64. There had been no intervening hematuria. His leukocyte count was normal. His hemoglobin was 10 gm.; hematocrit, 33. Bromsulfalein retention was 6 per cent at 45 minutes. Prothrombin value was 70 per cent of normal, whereas previous evaluations had been within normal limits. Chest x-ray was essentially unchanged. X-rays of the colon showed no evidence of recurrence of the pathologic condition of the colon and diverticula, which had previously been noted, were still present in the descending and sigmoid colon.

SUMMARY

1. Review of literature and this institution's experience with lymphosarcoma of the bowel is presented.

2. An illustrative case of lymphosarcoma of the small bowel and another of lymphosarcoma of the colon are presented.

3. The difficulties of early diagnosis are apparent.

4. The management of these problems needs further study and improvement.

5. The treatment of choice is surgical removal of the lesion followed by radiation.

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PRIMARY CYSTS AND TUMORS OF THE SMALL BOWEL MESENTERY

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Masses in the small bowel mesentery first attracted attention in 1507 when Benivieni of Florence[2] described at autopsy a cyst which he found originating in the small bowel mesentery. In 1880 Tillaux[9] reported the first successful removal of a mesenteric tumor. In 1897 Harris and Herzog[6] reported a collected series of 57 cases of solid mesenteric tumors; and Moynihan[7] reporting on mesenteric cysts that same year, estimated that about 100 cases of mesenteric cysts had already been reported in the world literature. Since that time, Rankin and Major,[8] Ackerman[1] and others have added to our information concerning such masses.

SOURCE OF MATERIAL

This present study of mesenteric masses is based upon 17 cases encountered at St. Francis Hospital, Evanston, during the period 1948 to 1959; of these 5 were cysts, and 12, tumors. This represents a patient incidence of 0.072 per cent in 23,579 general surgical admissions. During the period 1950 to 1959 at Illinois Research and Educational Hospitals, 4 mesenteric masses, all tumors, were encountered, a patient incidence of 0.037 per cent in 10,865 general surgical admissions.

ETIOLOGY OF MESENTERIC MASSES

Early investigators such as Dowd[5] and Carter[3] believed that embryonal rests or embryologic malformations were responsible for a large proportion of mesenteric masses, particularly cysts. However, Rankin and Major[8] and more recent investigators have been unable to substantiate the etiologic importance of such rests. Moynihan[7] believed trauma to be the usual etiologic factor in hemorrhagic cysts, but hemorrhage into a pre-existing cyst, when there is no history of trauma, is difficult to exclude. At present, one can only say that the principal origin of most mesenteric masses is neither from embryonic rests nor trauma, although these may occur, but rather from benign or malignant tumors arising in an otherwise normal small bowel mesentery.

In addition to cysts and tumors, mesenteric masses may be produced by aneurysms of the mesenteric vessels, although their differentiation from tumors or cysts should not be difficult. However, it would appear that, clinically, there exists a previously unmentioned group of cases which should be included in any classification of mesenteric masses, namely, cases in which the mesentery becomes grossly altered as a result of disturbed function of the intestine or mesenteric structures. Whipple's disease, intestinal lipodystrophy, which in one of our cases had transformed the small bowel mesentery into a tumor-like mass which was palpable abdominally, is an example of this group. Such a mass, although not a tumor or cyst, nevertheless requires differentiation from other mesenteric diseases, if errors in therapy are to be avoided. There may be other "physiologic diseases" capable of producing tumor-like alterations in the small bowel mesentery which would fall into such an etiologic classification although none were encountered in this series.

PATHOLOGIC CLASSIFICATION

An awareness of the pathologic possibilities presented by a primary mesenteric mass is of distinct value to the surgeon. It is evident any of numerous types of tissue including vascular, lymphatic, nervous, or connective tissue may be the source of a tumor or cyst. In addition, embryonic rests or grossly misplaced or duplicated adult tissues are a less frequent cause of a mesenteric mass.

The following outline lists the benign primary masses which have been reported previously in the small bowel mesentery. Those lesions which have been encountered at St. Francis or Illinois Research Hospitals are indicated by an asterisk. Rankin and Major[8] contend that lymphatic cysts are the most frequently encountered benign mesenteric mass; Courtney and Fala,[4] in a collective review of 216 solid tumors of the mes-

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I. Benign masses of the small bowel mesentery
  A. Cysts
    1. Lymphatic (chylous)***
    2. Enclusion (serous)**
    3. Traumatic (hemorrhagic)
    4. Enteric (mucoid)
  B. Solid tumors
    1. Lipoma*
    2. Fibroma
    3. Neurofibroma
    4. Ganglioneuroma
    5. Endothelioma
    6. Hemangioendothelioma*
    7. Dermoid
    8. Tetratoma — embryonal
    9. Desmoid (following trauma)*
    10. Papillary mesothelioma*
  C. Physiologic abnormalities
    1. Whipple's disease (intestinal lipodystrophy)*
    2. Aneurysms of mesenteric vessels

II. Primary malignant tumors of the small bowel mesentery
  A. Lymph nodes
    1. Hodgkin's disease**
    2. Lymphoblastoma**
    3. Small cell sarcoma*
    4. Reticulum cell sarcoma*
  B. Neurofibrosarcoma**
  C. Fibrosarcoma*
  D. Liposarcoma*
  E. Fibroliposarcoma*
  F. Myosarcoma*

entery, found lipoma to be the most common benign tumor.

This outline constitutes a list of the reported primary malignant tumors of the small bowel mesentery. The neoplasms reported in this series are indicated by an asterisk and are notable for their variety. Ackerman[1] stated that liposarcoma is the most frequently encountered malignant tumor of this area, although Stout[10] recorded fibrosarcoma to be the most frequent. Our experience, on the contrary, suggests that primary tumors originating in the mesenteric lymph nodes are much more frequent than either of the aforementioned tumors.

The patients with neurofibrosarcoma of the small bowel mesentery reported are of interest because the presence of the stigmata of neurofibromatosis on physical examination suggested the correct diagnosis of neurofibromatosis of the abdomen with malignant change before abdominal exploration.

* Indicates the number present in this series.

DIAGNOSIS

As illustrated in figure 1, the center of masses originating in the small bowel mesentery is near the umbilicus along a line running from the left upper quadrant to the right lower quadrant which approximates the root of the small bowel mesentery. Some degree of mobility is usually present, especially in the transverse direction, unless there has already been invasion of the retroperitoneal tissues causing fixation. The age of the patient is of little aid in making a diagnosis since, in our patients, the age of patients with benign tumors varied between 6 and 71 years with an average age of 45 years, whereas the age of patients with malignant tumors varied between 26 and 71 years, with an average age of 57. Moynihan[7] has stated that benign cystic disease of the mesentery is more common in women than men; 4 of our 5 patients with cysts were women. Malignant mesenteric disease, in our experience, was three times as frequent as benign mesenteric disease in men, whereas in women, only 42 per cent of the mesenteric masses were malignant.

Table 1 compares the duration and incidence of the symptoms and signs in benign and malig-

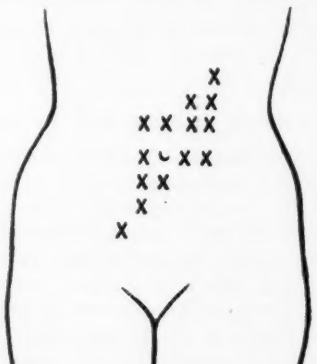

FIG. 1. The center of the 14 primary mesenteric masses as located by palpation. Since these masses arise in the mesentery, their centers roughly correspond to the line of mesenteric attachment to the posterior abdominal wall. In the absence of retroperitoneal invasion with fixation, these masses therefore, are mobile, particularly in a transverse direction.

TABLE 1

The relative incidence of symptoms and signs in mesenteric disease (21 patients)

Symptom or Sign	Malignant* (11 Patients)	Benign† (10 Patients)
	per cent	*per cent*
Abdominal mass	81.8	50.0
Abdominal pain	54.6	60.0
Weakness	54.6	30.0
Fullness	54.6	60.0
Weight loss	45.5	30.0
Diarrhea, Cramps, Anorexia, Vomiting	45.4	30.0

* Duration of symptom: range, 0.5 to 36 months; average 14.5 months.

† Duration of symptom: range, 0.5 to 36 months; average 11 months.

nant disease but, unfortunately, no useful differences are evident. An abdominal mass was felt in over 80 per cent of the patients with malignant disease; but in only 50 per cent of those with benign mesenteric disease. Abdominal pain was present in about two thirds of all patients, but weakness, fullness and weight loss was much more frequent among those patients with malignant disease. Diarrhea, cramps, anorexia or vomiting was present in only 30 per cent of the patients with benign disease, but in over half of those with malignancy.

DIAGNOSTIC PROCEDURES

Mesenteric masses, because they are located outside the gastrointestinal, biliary, or renal systems, are visualized by x-ray only when they produce demonstrable pressure on these systems. Such findings were evident in only 2 of our 16 patients. In one, the stomach and colon were displaced upward, and in another the small bowel showed compression by an extrinsic mass. Intravenous pyelography in no case showed ureteral displacement or compression. In none of these cases was ascitic fluid available for study. Routine blood, urine, and chemistry studies were relatively uninformative in both benign and malignant cases. Because of the scarcity of definitive findings in mesenteric growths, other than those of a movable mass, the correct diagnosis of such masses is usually made by exclusion and confirmed only by surgical exploration.

TREATMENT

All mesenteric masses should be removed, if possible, because even the benign lesions eventually cause pain and compression of neighboring structures. Benign mesenteric masses can often be removed by local excisions or the sacrifice of only small segments of intestine. Of our 10 benign cases of mesenteric disease, removal was possible in all but 2; one of these inoperable cases was an immense lipoma; the other patient had Whipple's disease. Removal in both of these instances would have been impossible without sacrifice of the entire small bowel blood supply. Malignant tumors which do not involve indispensable structures or the removal of an amount of gut incompatible with life should be resected. However, of the 11 cases of malignant tumors in this series, only 4 were resectable, a neurofibrosarcoma, a fibrosarcoma, a liposarcoma and a fibroliposarcoma. Of these four patients, only the patient suffering from fibroliposarcoma survived more than 5 years; he is free of disease 10 years after his surgery.

SUMMARY

Primary masses of the small bowel mesentery are uncommon and constitute between 0.037 to 0.073 per cent of general surgical admissions.

Benign and malignant mesenteric masses occur with roughly equal frequency.

All mesenteric masses should be removed, if possible, since their growth produces pain and compression of neighboring structures even if benign.

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HYPERTROPHIC PYLORIC STENOSIS: A SURGICAL AND PATHOLOGIC REPORT OF AN APPARENTLY SUCCESSFUL MEDICALLY TREATED CASE SOME 18 MONTHS LATER

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The majority of papers advocating medical therapy of hypertrophic pyloric stenosis have appeared in the Scandinavian and British literature. Such studies as those of Malmberg,[7] Rinvik,[10] Dobbs,[3] Svensgaard,[12] and Elgenmark[4] have expressed the superiority of medical treatment.

The present case illustrates a possible pitfall in the medical treatment of congenital pyloric stenosis.

CASE REPORT

L. L. R. (CGH 120981), an 18-month-old first born white boy was admitted to the Colorado General Hospital on July 5, 1959, with the history that he had swallowed a penny 4 weeks before admission. The patient was followed by his private physician, during which time he was treated with Pathilon, an anticholonergic, for 3 weeks without passage of the penny beyond the pylorus, as shown by x-ray. The patient was then referred to the Colorado General Hospital for surgery.

Past history revealed that at 4 weeks of age, the patient had a severe bout of vomiting and constipation. This persisted approximately 1 week and, at that time, a diagnosis of pyloric stenosis was made. During this time, the patient was treated with antispasmotic drugs and frequent small feedings. The vomiting lasted approximately 1 week and the child had had no further symptoms.

Family history and review of systems were noncontributory except to note that there were no other known cases of pyloric stenosis in the family.

Physical examination on admission revealed a well developed, well nourished 18-month-old child whose only positive physical finding was that of a slightly hyperemic pharyngeal mucosa.

Laboratory data on admission revealed leukocytes of 11,300 which, when repeated the next day, was 6650. The urinalysis was within normal limits.

X-ray examination with barium contrast material revealed the presence of a coin in the stomach (fig. 1). During this examination, the duodenum was never seen well because the barium was retained in the stomach for over two hours. A repeat examination two days later was strongly indicative of pyloric hypertrophy, possibly with a spastic component.

On the fourth hospital day, the patient was taken to the operating room where, under cyclopropane and ether anesthesia, a 6-cm. paramedian right upper quadrant muscle-splitting incision was made. The coin was palpated in the stomach. The pylorus was 2 cm. long, firm and hard (fig. 2). A longitudinal incision through the muscularis to submucosa, 2.5 cm. in length, was made from the pyloric vein extending proximally over the stomach.

A gastrotomy incision was then made and the penny extracted. A large Kelly clamp was introduced through the pylorus and it appeared to spread easily. The gastrotomy incision was closed, appendectomy performed, after which the abdomen was closed in layers.

The patient was placed on penicillin and streptomycin postoperatively because of a rectal temperature of 38.5°C. This rapidly fell to normal. On the evening of the first postoperative day he was started on glucose water feeding, and by the fourth day was taking a soft diet well. He was advanced to a regular diet on the fifth day and sent home afebrile on the sixth.

The patient has been seen frequently during the 7 months since surgery and has been asymptomatic.

The history of pyloric stenosis is well documented from 1627 up to the present time.[6] The development of medical therapy has been reviewed.[1, 2, 5, 6, 8, 9, 11]

SUMMARY

This case presentation illustrates a subsequent complication in a patient with a diagnosis of hypertrophic pyloric stenosis treated medically. Although asymptomatic, surgical intervention was indicated to remove a swallowed penny. Typical pyloric stenosis was seen and treated surgically when the penny was removed. The

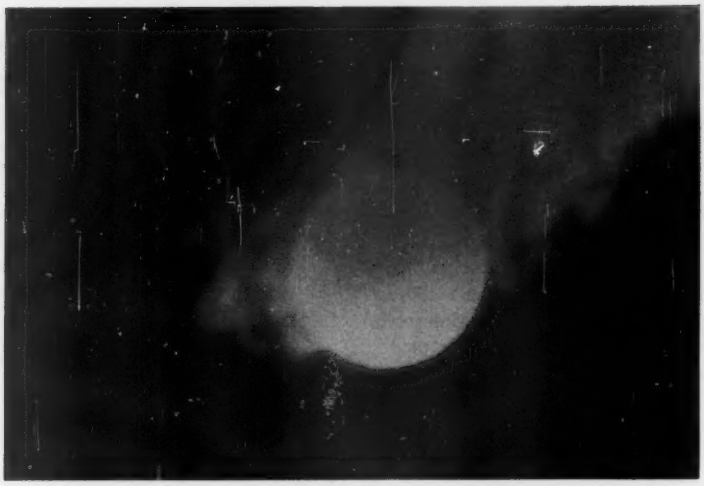

FIG. 1

FIG. 2

pathologic condition apparently persists for many months after symptoms have disappeared in this disease.

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RADIATION INJURY TO THE RECTUM AND SIGMOID COLON

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Radiation injury to the colon is an accepted well known hazard encountered with irradiation therapy for pelvic cancer. The rectum and sigmoid colon are especially vulnerable to injury because of their proximity to the uterus and cervix, and due to the fact that the intestine is about three times as sensitive to irradiation as is the uterus and cervix.[5] This sensitivity is variable and unpredictable.[13] It accounts for severe reactions secondary to minimal irradiation.[4]

Buie and Malmgren[4] described the rectal damage as being a factitial proctitis. They found that rectal symptoms occurred in 65 of 2073 (3.13 per cent) patients having pelvic lesions treated by irradiation. Aune and White[2] reported that 3.4 per cent of 670 patients who received irradiation therapy for cancer of the cervix developed gastrointestinal complications. The true incidence depends upon the accuracy and vigilance in follow-up examinations. Aldridge[1] reported on 189 cases of uterine carcinoma treated by irradiation therapy and followed with proctoscopic examinations. Rectosigmoidal complications occurred in 16.9 per cent. He felt that this incidence was high because previous reports had included only the more severe cases. Accurate appraisal is possible only after a follow-up period of five years or more.[14]

Discussion of different methods of irradiation therapy for pelvic cancer is not the subject of this report, but therapy usually consists of combined complementary deep x-ray and radium irradiation. The comparative amounts of each vary, and it is known that irradiation damage can occur with the use of either type of irradiation. Fabrikant and co-workers[8] reported on 71 cases of irradiation injury to the intestines. Radium only had been used in 9.8 per cent of the cases; x-ray only in 12.6 per cent; and a combination of the two in 67.8 per cent of the cases. The introduction of the cobalt unit has made it possible to administer an increased amount of deep irradiation with more accuracy and less skin damage. What effect this will have upon adverse intestinal damage is conjectural. A case of rectosigmoidal irradiation damage secondary to radium and cobalt therapy for carcinoma of the cervix is presented.

CASE REPORT

A 36-year-old white woman was diagnosed as having carcinoma of the cervix on August 19, 1957. On August 22 she was admitted to the hospital. Examination revealed a large fungating tumor of the cervix with infiltration into the left parametrium. One-hundred mg. of radium were applied in an intrauterine brass T applicator for 30 hours. The tumor responded to treatment and on September 13, 1957, 100 mg. of radium were again applied for 24 hours. Fifty mg. were in an intrauterine brass T applicator, and 25 mg. in a bomb applied to each fornix. Cobalt therapy was given in 26 treatments between August 26, 1957, and September 27, 1957. The central area was shielded and irradiation given through two portals in the anterior posterior direction. The total irradiation received was 6700 gamma r of radium at point A (where the ureter and the uterine artery cross); 1700 gamma r of radium and 4000 gamma r of cobalt therapy for a total of 5700 gamma r at point B (lateral pelvic wall).

In April 1958, approximately 7 months after completion of irradiation therapy, the patient began to have cramping abdominal pain associated with tenesmus and the passage of dark blood clots mixed with the stool. There was no diarrhea. On June 10, 1958, she was admitted to the hospital and examination revealed an induration in the cul de sac of Douglas and in the pelvic adnexa. There was no gross evidence of local recurrence of the cervical tumor. The anterior abdominal wall was indurated.

Sigmoidoscopic examination revealed hyperemia of the rectum and rectosigmoid area of the colon. The mucosa was granulomatous. The mucosa bled easily on touch. There were numerous areas of telangiectasis and ulceration. There was evidence of stricture formation in the lower sigmoid. The findings were those of radiation proctitis with ulceration and stricture formation.

The vital signs were normal. Urinalysis normal. Complete blood count showed a hemoglobin of 11.8 gm. (77 per cent), erythrocyte count 4.1

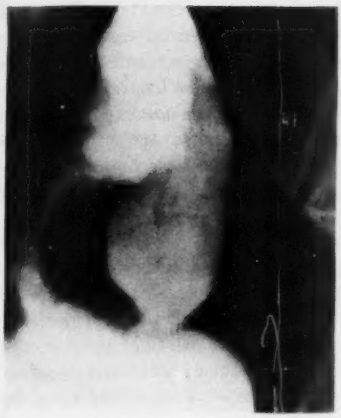

FIG. 1. Roentgenogram revealing an area of narrowing in the rectosigmoid region, approximately 6 cm. in length.

million, leukocyte count 8200 with a normal differential. A barium enema showed a narrowing of the rectosigmoid colon approximately 6 cm. in length (fig. 1). The bowel was prepared with intestinal antibiotics and on June 19, 1958, surgery was performed through a lower midline incision. There was no evidence of local extension or distal metastasis of the cancer. Partial obstruction of the sigmoid colon was noted. The uterus was adherent to the rectosigmoid and there was induration of the perirectal area. The sigmoid was divided six inches above the strictured area preparatory to resection. During dissection the colon spontaneously disrupted through an area of necrosis. The necrotic area was excised and the lower rectal stump closed. An end-type sigmoid colostomy was performed through a stab wound in the left lower abdomen and the ovaries were removed.

Microscopic examination of the tissue revealed an ulceration and necrosis extending through the bowel wall into the adjacent fibrofatty tissue. There was extensive edema and acute inflammation present.

The patient had an uneventful postoperative recovery and was discharged July 2, 1958. Follow-up examinations were done with no evidence of recurrence of the carcinoma or further intestinal bleeding. On January 24, 1960, 19 months later, she was readmitted to the hospital. Sigmoidoscopic examination showed minimum evidence of radiation proctitis in the rectal stump. The bowel was prepared with intestinal antibiotics and cleansing enemas. On the morning of surgery, January 27, cystoscopy revealed a normal appearing bladder. Ureteral catheters were inserted on both sides to facilitate identification of the ureters during surgery. A rectal tube was inserted to aid in identification of the rectal stump. The abdomen was explored through a midline lower abdominal incision and again there was no evidence of residual carcinoma or nodal metastasis. Extensive adhesions about the rectal stump lying in the cul de sac were divided and the stump opened. The bowel wall of the stump was thickened and petechia were noted on the mucosa. The colostomy was dissected free and mobilized sufficiently for end-to-end anastomosis of the sigmoid to the rectal stump. The pelvis was irrigated and drained through the previous colostomy site. A normal appendix was removed.

The patient did well postoperatively and was discharged on February 10, 1960, with slight drainage from the drain site. She was eating well and having regular bowel movements. She has continued to do well and is free of symptoms.

COMMENTS AND DISCUSSION

Onset of symptoms in this case occurred about 7 months after irradiation therapy. Buie and Malmgren[4] found that the average onset of symptoms was at 4.75 months. Symptoms may begin immediately or occur as late as 9 years after therapy.[2, 4, 7, 8, 15]

Symptoms of abdominal pain, tenesmus and the passage of blood from the rectum are typical in these injuries although the character of the symptoms vary and change with the type and extent of intestinal pathologic condition. Stages of advancement in pathology have been classified.[1, 3, 5] In stage 1 there is congestion, edema and hyperemia of the bowel usually involving the anterior rectal wall at the level of the cervix. The entire circumference of the bowel may be involved. Telangiectasis is seen.[4] The vulnerable location of the rectum in relation to the cervix accounts for the frequency of damage to the midrectum.[7] A granulomatous mucosa occurs with increased severity of the proctitis. In this stage bleeding with the passage of mucous occurs due to trauma to areas of telangiectasis. Diarrhea is common as an early symptom.[8] Pain occurs later with progression in severity of proctitis. Also it has been observed that these gross pathologic changes may be seen without associated manifest symptoms.[14]

In stage 2 ulceration occurs; again this usually occurs on the anterior rectal wall at the level of the cervix. The predominant symptom is an increasing severity of pain, especially in association with bowel movements.[7]

Stage 3 is characterized by stricture formation which may be aggravated by edema and spasm.[7] There is partial to complete bowel obstruction with cramping abdominal pain, bleeding and constipation.

In stage 4 there is bowel perforation, rectovaginal or vesicovaginal fistula formation.

Although classification of pathology and associated symptoms are convenient for discussion, it should be noted that there is not always an orderly progression of intestinal damage. Todd[15] followed cases receiving irradiation therapy by routine proctoscopic examination. He found that most of the cases with acute proctitis eventually subsided without progression to the later chronic type damage. Also it was interesting to note that chronic reactions could occur with no preliminary early evidence of acute proctitis. Todd described two types of chronic reaction secondary to the use of radium. One was an intrinsic reaction in which the pathology was limited to the rectum. The other was an extrinsic reaction in which there were both rectal and perirectal tissue pathology. He suggested that the intrinsic type lesion is the result of direct irradiation damage whereas the extrinsic reaction was related to a decrease in the vascular blood supply to the perirectal tissue and rectum secondary to irradiation arteritis and thrombosis in the smaller hemorrhoidal vessels. This results in necrosis and massive perirectal fibrosis with fixation of the rectum. Intrinsic and extrinsic reactions are not distinguished on sigmoidoscopic appearance alone, although stricture formation in the extrinsic variety is accompanied by fixation and distortion of the bowel with extreme difficulty in passing the sigmoidoscope. It is felt that there were both types of reaction in the case presented. The intrinsic lesion of necrosis and fibrosis with stricture of the rectosigmoid colon was associated with fixation of the rectum at a lower level with a partial obliteration of the cul de sac due to the extrinsic reaction.

It is the extrinsic reaction that may lead to confusion in diagnosis with erroneous conclusions that the pathology represents a primary carcinoma of the rectum or a recurrence with extension or metastasis from the previous cervical cancer. A hopeless outlook or the advocation of further irradiation therapy would be detrimental to the patient. Craig and Buie[7] found that 19 of 200 cases (9.5 per cent) of irradiation damage had been referred to the Mayo Clinic with the clinical diagnosis of rectal carcinoma. Fourteen had been told that the original pelvic cancer now involved the bowel and five had been diagnosed as having primary rectal carcinoma. It is of paramount importance to make the correct diagnosis. All diagnostic means including transsigmoidoscopic biopsy should be used if necessary for differentiation.[10, 19]

Management of these lesions depends upon the stage of pathology. In general, acute proctitis (stage 1) usually subsides spontaneously or responds to a bland or low residue diet, sedation, antispasmodics and intestinal antibiotics. This is used in conjunction with cleansing enemas using warm normal saline. The enemas are given after each bowel movement and are followed by rectal instillation of olive oil, cod liver oil, starch or bismuth solution.[3-8, 19] Sherman[14] has instilled hydrocortisone in a water soluble jelly base obtaining very satisfactory results. Similar treatment is recommended for ulceration with proctitis (stage 2). During this treatment the patient must be under careful observation because of the possibility of complications of severe bleeding, intestinal obstruction or perforation of the bowel.[18] Buie and Malmgren[4] stated that complete healing never occurs in less than 14 months and usually takes two years. Nevertheless, Sherman[14] found that "the majority of the grade 1 and 2 lesions have largely disappeared in approximately two months after a regime of topical administration of hydrocortisone to the lesion." Colcock and Hume[6] feel that if there's no significant improvement after 2 to 3 weeks of treatment then a proximal colostomy should be done. Other indications for surgery are the complications of severe bleeding and intestinal obstruction or perforation. The management of these complications are not discussed.

In stricture of the rectum (stage 3) conservative treatment may be tried in the absence of intestinal obstruction when the stricture can be reached by the sigmoidoscope and gradual gentle dilation done. In case studies by Aldridge[1] symptoms were relieved in 18 of 29 cases with stricture when nonoperative treatment was used. Similarly, others[13, 17] have been successful, although Sherman found that only the early strictures responded to conservative treatment. In the face of obstruction a transverse colostomy is performed. Successful dilation following this

may allow eventual closure of the colostomy without further surgery.[5] However, closure of the colostomy or definitive resection of the stricture should be delayed until a sufficient length of time has elapsed to permit healing. It is prudent to wait 18 to 24 months for definitive surgery. During this time one is also able to determine possible recurrence or metastasis of malignancy which may affect the decision as to definitive surgery. Because of the basic pathology of arteritis, thrombosis and diminished circulation to the bowel, one must take care in preserving the remaining blood supply to the rectum when performing an excision of the stricture with anastomosis. On occasion, complicating fistulas have followed anastomosis probably because of inadequate blood supply or failure to wait a sufficient length of time allowing stabilization of the disease with healing.

In the case presented definitive end-to-end anastomosis was delayed for 19 months. At the end of this time there was minimum evidence of intrinsic disease although perirectal fibrosis was considerable. The blood supply to the rectal stump was diminished, but it was felt that the circulation was adequate for successful anastomosis. No complications occurred. The use of the ureteral catheters were of considerable benefit in permitting accurate localization of the ureters during dissection.

The treatment of vesicovaginal and rectovaginal fistulas (stage 4) will not be discussed in this paper.

In conclusion, the question arises as to the significance of the influence of radioactive cobalt as a causative factor in radiation necrosis of the bowel. Cases presented by Todd of both intrinsic and extrinsic lesions of the bowel were all secondary to radium therapy alone. Nevertheless, he suggested that his experience with the increased prevalence of the extrinsic type reaction suggested that widespread pelvic involvement was possibly associated with the fact that deep x-ray therapy was being used more and more. Twombly and associates[16] compared the incidence of rectal damage in four series of cases in which the amount of radium therapy was constant at 4500 mg./hour. With an increase in the combined x-ray therapy from 3240 r to 14,400 r the incidence of rectal damage increased from 5.5 per cent to 11.8 per cent. Peighthal[11] found 13 instances of major intestinal damage in 100 cases of carcinoma of the cervix treated by both radium and high voltage therapy. He compared this with 62 other cases in which almost double the amount of radium but only one-half the amount of high voltage therapy was used, resulting in only two bowel injuries. He concluded that intestinal injuries were usually related to the use of high voltage irradiation. Reeves[13] stated that there may be some increase in radiation damage in using supervoltage and cobalt[60] therapy for carcinoma of the cervix. It is probable that irradiation necrosis of the bowel is secondary to the combined effects of radium and x-ray irradiation and is related to overdosage to the sensitive intestinal tract. Certain mechanical factors, such as positioning of the radium applicators, the position of the uterine fundus, the normal anatomic proximity of the rectum to the cervix and the presence of adhesions holding small or large bowel in a constant position within radiation range, all contribute toward possible irradiation overdosage to the intestine and particularly the rectum and sigmoid colon.[1, 10, 15] Nevertheless, the primary objective of destruction of cancer tissue with prolongation and conservation of life is paramount in irradiation therapy. The hazards associated with therapy are considered acceptable and of secondary importance, although every effort is being made to prevent damage to normal structures and at the same time maintain the highest possible cure rate in the treatment of cancer of the cervix.

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